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This number deals principally with peripheral vascular disorders which have proved to be of special importance in wartime. Professor J. R. Learmonth, who contributes the introductory article, has occupied the Chair of Surgery in the University of Edinburgh since September 1939. He was previously Regius Professor of Surgery in the University of Aberdeen. He has charge of a Nerve Injuries Unit and Peripheral Vascular Unit in an Emergency Medical Service hospital under the Department of Health for Scotland, where many patients suffering from peripheral vascular disorders have been concentrated during the war.

Dr. William Blackwood has been Pathologist to the Scottish Mental Hospitals Laboratory, the Royal Infirmary, Edinburgh, and Honorary Lecturer in Neuropathology in the University of Edinburgh since 1939. Work on immersion foot arose out of these contacts, and from a meeting with Surgeon Commander C. C. Ungley, after he had sent a portion of nerve for biopsy to Dr. Blackwood's predecessor, Squadron-Leader A. C. P. Campbell.

Dr. R. L. Richards, a graduate of Aberdeen University, has since the summer of 1940 been assistant at a Scottish Nerve Injuries Unit in an Emergency Hospital under the Department of Health for Scotland. He has been making a special study of the vasomotor disorders which result from injuries of peripheral nerves. Work on immersion foot has been carried out in collaboration with Surgeon Commander C. C. Ungley at a naval hospital in Scotland and at the Nerve Injuries Unit directed by Professor Learmonth.

Mr. B. C. Maybury is Surgeon to St. Thomas's Hospital, London, where he was earlier a pupil of Sir George Makins and Sir Cuthbert Wallace—two pioneers of military surgery. Mr. Maybury was for nearly three years during the war of 1914–18 a surgical specialist at a Casualty Clearing Station, and also spent a short time at a Base Hospital. He has had considerable personal experience of arterial injuries and has long had a special interest in this subject.

Mr. S. M. Cohen is a South African surgeon who joined the British Emergency Medical Service at the outbreak of war, and has thus gained experience of the treatment of war casualties. He has made a special study of traumatic arterial spasm, and chose this as the subject for his lecture on election in 1942 as Hunterian Professor of the Royal College of Surgeons of England.

Professor Learmonth, Mr. Cohen, and Mr. Maybury are members of a Subcommittee on Vascular Injuries which was constituted early in 1944 by the War Wounds Committee of the Medical Research Council.

In the planning of this number of the Bulletin Professor Learmonth has given much personal advice and assistance. In addition, he and his Edinburgh colleagues, Dr. Blackwood and Dr. Richards, have contributed many of the reviews of selected papers.

SPECIAL CONTRIBUTIONS

PERIPHERAL VASCULAR DISORDERS

J. R. LEARMONTH, CH.M., F.R.C.S.ED.

The pathological conditions dealt with in this issue of the *Bulletin* have this in common, that in all the possibility of ischæmia is present. This may arise without any breach of the arterial tree by external forces; on the other hand, when an artery is wounded, in addition to the complete or partial interruption of the conduit supplying blood to a part, there will be hæmorrhage, internal, external, or both, and thus any tendency to ischæmia as a result of the loss of one (or more) of the transmitting channels may be accentuated by a reduction in the total volume of the circulating blood. These considerations suggest the following clinical classification of the affections which may produce ischæmia:

A. *Resulting from Injury*
1. Contusions of arteries: (*a*) with thrombosis (*b*) without thrombosis
2. Arterial spasm: (*a*) after open injuries (neighbourhood gunshot wounds) (*b*) after closed injuries (fractures, crush injuries)
3. Wounds of arteries: (*a*) complete (*b*) partial
4. Arterial hæmatomata or "false" aneurysms
5. Arteriovenous fistulæ
6. Traumatic arteritis
7. Traumatic venous thrombosis (or venous spasm)
8. After division of peripheral nerves

B. *Occurring in Organic Vascular Disease*
1. Syphilitic arteritis
2. Arteriosclerosis
3. Diabetic arteritis
4. Thrombo-angiitis obliterans

C. *From Local Effects of Cold*
1. The Raynaud phenomenon, with or without nutritional changes
2. Immersion foot
3. Frostbite

Some of these conditions are dealt with in detail in individual articles elsewhere in this issue; but it cannot be too strongly emphasised that the rational treatment of all of them depends on attention to two fundamental principles: the control of hæmorrhage when present, and the maintenance of tissue nutrition.

Control of Hæmorrhage

In war surgery the control of bleeding is a problem of far greater magnitude and urgency than in civil surgery; yet it is a curious reflection that in spite of the wealth of material, only one major improvement in this technical matter has emerged directly from military experience—the reintroduction of the ligature by Paré about the middle of the 16th century. In the first two centuries of the Christian era, both Celsus and Galen employed the ligature. The former divided the bleeding vessel between ligatures placed on each side of the vascular wound; the latter raised the artery on a hook, and, in his own words, "it is safer for the operator to put a double loop round it and to divide between, and let these ligatures be of a material which will not readily decompose. Such a material . . . can be got from the Gaietans, who bring it from the country of the Kelts" [? linen]. Galen also suggested catgut: "If you cannot get this" [linen], "choose the material least liable to decompose . . . such as fine catgut." In mediæval times the cautery and boiling oil were preferred to the ligature, until in 1552 Paré reintroduced it, after catching the bleeding parts in a crude forceps. Thereafter the ligature kept its place in operative surgery, although suppuration and consequent secondary hæmorrhage compromised its efficacy, and its use was firmly established by John Hunter in 1785, in the treatment of popliteal aneurysm.

It was natural to seek for an absorbable ligature, which would not promote suppuration by acting as a foreign body. In 1814 Physick used untanned buckskin; in 1818 Astley Cooper employed catgut. Crude catgut, as was to be learnt later, contains bacteria; and after numerous surgeons had tried many different kinds of thread, Lister in 1868 returned to catgut, which he sterilised with carbolic acid. In 1881, he produced catgut hardened by chromic acid to resist absorption for some time, a method of preparation to which Macewen also contributed. Sterilised and hardened catgut has remained the most widely used absorbable ligature. With the advent of modern aseptic operative technique, and the practice of sterilising other forms of thread by boiling, many surgical schools have returned to the unabsorbable ligature of silk, linen or cotton, particularly in aseptic cases.

The general use of the ligature brought with it the need for some method of isolating and temporarily occluding bleeding vessels, and the crude pincers of Paré paved the way for the artery forceps, originally designed by Spencer Wells in 1872, and subsequently modified in ways too numerous to mention, according to the purposes to which it was to be put and the preferences of individual surgeons. So, too, the tourniquet has been modified to meet the needs and gentler methods of modern surgery, although for "first-aid" it persists in its primitive form—introduced by Morel in 1674—of a band of cloth tightened by turning a stick. However useful the tourniquet may be in an emergency, it is to be regarded as a purely temporary substitute for more exact methods.

The local repair of wounds of blood vessels was evolved from the experimental work of Carrel & Guthrie, 1905, from which emerged techniques for the closure of wounds of vessels, and later for the restoration of vascular continuity by axial anastomosis. These methods also opened up the possibility of conservative treatment of arteriovenous fistulæ. Their applicability in war surgery is limited because they are available only in favourable conditions including both absence of infection from the wound and proper operative facilities.

Maintenance of Tissue Nutrition

In contrast with the lack of any revolutionary advance in technical methods of controlling bleeding, much more progress has been made in solving the diverse problems of maintenance of tissue nutrition: these advances may be considered in turn.

i. *Restoration of blood volume:* Inadequate circulation resulting from gross reduction in the volume of circulating blood has been dealt with by the perfection of techniques for infusing blood or blood-products.

In Britain the scientific investigation of the possibilities of transfusion dates from the work of the great architect Sir Christopher Wren in 1657, and soon engaged the attention of the newly formed (1661) Royal Society. For two and a half centuries sporadic attempts were made to utilise clinically a procedure at once attractive and somewhat dramatic; but technical difficulties, and the violent and sometimes fatal reactions which followed the transfusion of either animal or human blood, set a limit to its justifiable use until the beginning of the 20th century, when the explanation of at least some of the reactions after transfusion evolved from the discovery of isoagglutination, and the subsequent reduction of human blood into groups (Landsteiner, 1900, 1901; Janský, 1907; Moss, 1910) which provided a basis for the choice of a compatible donor. For some time thereafter, direct transfusion, or transfusion from a vessel coated with paraffin wax (Kimpton & Brown, 1913) were used; but these methods, the former inaccurate, the latter involved, were soon displaced by the discovery (Agote, 1915; Lewisohn, 1915) that sodium citrate was an effective and non-toxic anti-coagulant, and on November 14th, 1914, the first transfusion by this method—so soon to be of cardinal therapeutic importance in the war of 1914–1918—was performed by Agote of Buenos Aires. In the years after the war, transfusion of blood rapidly extended its proven value, and ultimately supplies of donors were organised throughout Britain on a voluntary basis. About 1936 American workers found that it was possible to store citrated blood in blood-banks; and when in 1938 it became clear that large amounts of blood might be needed for casualties, the *Medical Research Council* undertook the establishment of blood supply depots around London, and has since supported and co-ordinated clinical and research work on transfusion in Britain. In Scotland the Department of Health sponsored the foundation of the Scottish National Blood Transfusion Association to co-ordinate and extend transfusion work in Scotland; in England the Ministry of Health, by direct action, organised transfusion services throughout the provinces. A similar "bank" was also set up by the Army Transfusion Service.

The discovery that plasma could be used to restore blood volume was not long delayed; the value of blood-banks was greatly enhanced, for plasma keeps much longer than stored blood, from which it can be recovered; and it may be given without previous tests for compatibility. Serum was found to have similar properties. Finally the problems of storage and ultimately transportation were greatly simplified by the elaboration of techniques for drying plasma and serum, founded on the work of Flosdorf & Mudd (1935) in America and the extension of this by Greaves & Adair (1936) in Britain; when needed, they were reconstituted on the spot by the addition of sterile distilled water or physiological saline. Liquid plasma and serum are unstable and tend to precipitate slowly during storage; dried plasma is stable but involves the transport of two bottles instead of one. Hence attempts have been made to produce a stable concentrated solution, of which the most successful is that of Cohn and his associates in America. This involves the alcohol fractionation of plasma proteins and the use for transfusion of the albumin only, in the form of a 30 % solution (various globulin fractions being used for other purposes). Attempts to dispense completely with the human donor have led to trial of other proteins in solution—bovine plasma albumin (as such or after treatment to remove species specificity), gelatin, isinglass, etc. The consensus of opinion at present favours human plasma proteins, and in Britain difficulties of supply rule out the large-scale preparation of albumin.

All these advances in procedure have been highly developed in Britain, and so well have transfusion services been organised that they are available to the wounded—whether in battle or in civil air raid—during the earliest phases of treatment; a magnificent achievement which not only reduces immediate mortality but also brings the possibility of operative surgery to a high proportion of wounded.

ii. *Restoration of flow through the injured artery:* After wounds of major vessels, the ideal is to restore the flow of blood through its previous channels, or at least to keep up the flow temporarily until an adequate collateral circulation is established. The latter procedure has been attempted by the substitution of various types of cannulæ (Tuffier, 1915; Blakemore, Lord & Stefko, 1942) for the normal conduit, and has the theoretical advantage of availability at an early stage of treatment. The former involves the suture of arterial wounds, the anastomosis of severed arteries, and the closure of fistulæ between arteries and veins, techniques which demand a much more elaborate operative setting and therefore deferment to a later stage of treatment. In the past, results of both types of procedure have been vitiated by the occurrence of thrombosis within cannulæ or at suture lines; but within recent years the discovery of anti-coagulant substances (heparin—Howell, 1916; Charles & Scott, 1933: dicoumarin—Campbell & Link, 1941; Butt, Allen & Bollman, 1941) has provided means by which, under adequate laboratory control, this complication may be prevented or minimised. Many such conditions (traumatic aneurysms, fistulæ) are best dealt with after an interval, and vascular centres have been set up, both in the battle areas and in Britain, where they can be dealt with under the most advantageous conditions.

iii. *Surgery of the sympathetic:* The development of the surgery of the sympathetic system has provided a method of modifying the circulation in an extremity. From a surgical point of view, the story begins with Claude Bernard who, in 1852, described the raised cutaneous temperature and increased fullness of subcutaneous vessels in the ear of the rabbit, after section of the ipsilateral cervical sympathetic trunk; then Brown-Séquard showed that stimulation of the trunk was followed by vasoconstriction. In the latter half of the 19th and the beginning of the 20th century the work of Gaskell, Langley and their school solved many problems in the anatomy and physiology of the sympathetic system, and by 1921 the work of Langley, Elliot and Cannon on sympathetic mechanisms, and Dixon, Loewi and Dale on parasympathetic mechanisms, had identified the humoral substances concerned in the mediation of nervous impulses traversing these systems.

In 1913 Leriche utilised as a therapeutic measure the temporary vasodilatation which follows the removal of the adventitial coat of the main artery of an extremity; but it was not until 1924 that Hunter and Royle noted the increased warmth which followed sympathetic ramisectomy in man. Since that date the operative technique of sympathectomy designed to modify circulation has been modified and revised many times. In Britain fundamental work of this kind has been done by Gask, Telford and Ross, while in the United States Adson and J. C. White have made notable contributions. White has also standardised methods of blocking the paravertebral sympathetic chains by the injection of procaine and alcohol. On the clinical experimental side, the papers of Sir Thomas Lewis and his school have thrown much light on vasomotor conditions, and have supplied not only a wealth of observations but also simple methods of studying such cases at the bedside and in the laboratory. The "warbaby" of this group, immersion foot, has found admirable British expositors in Ungley, Blackwood and Critchley.

It seems clear, from the work of Theis (1933) that, in the experimental animal, division of the appropriate sympathetic nerves will increase the total input of blood into a limb. Both in animals and in man, most of the increased flow traverses the subcutaneous vessels, muscle-tissue profiting less; but experience at vascular centres, both in Britain and abroad, supports the view that when ligation of a main artery in an extremity is inevitable, the risk of massive gangrene can be reduced by a preliminary sympathectomy, which allows a proportion of alternative vascular channels to dilate without the impediment of constrictor tonus imposed by their

extrinsic nerves. It is also possible that, until the casualty reaches suitable operative facilities, the same advantage may accrue, although temporarily, from paravertebral blocking by local anæsthetics of the appropriate sympathetic trunks.

iv. *Reduction of local metabolic rate :* It is a physiological commonplace that the direct application of heat to a part increases its metabolism, and therefore its demand for blood. When the part is ischæmic, a proportion of its impoverished blood supply is diverted by local heating to provide for increased local metabolism, and the total input of blood may ultimately be inadequate to maintain merely the vitality of the tissues, with the result that they die, and more or less extensive portions become gangrenous. Largely as a result of the teaching of Sir Thomas Lewis, the practice of heating locally parts threatened by gangrene has been abandoned ; thus the ischæmic limb of the arteriosclerotic or diabetic patient is to-day not deprived of its chances of recovery by the application of hot water bottles or the heat cage. Indeed the modern trend is actively to cool an ischæmic extremity, as tissue metabolism may be reduced by the local application of cold, and at a lower level of tissue metabolism there is a corresponding reduction in the threshold blood supply required to maintain vitality. The therapeutic application of cold in the treatment of threatened gangrene has been studied by Allen (1941 and later), who showed experimentally that when an extremity was excluded from the general circulation by a tourniquet, the period over which tissue remained viable was extended by cooling the limb to a temperature of about 5° C. Moreover, this lowered temperature also put an effective brake upon the multiplication and invasiveness of bacteria. These two effects of chilling have an obvious application in war wounds, especially in cases in which arterial injuries leading to a reduction in the circulation of a limb may be disastrous as a result of the rapid spread of anaerobic and aerobic infections ; and the principle has been put to material use in the present conflict.

v. *Prevention and management of lesions due to cold :* Nutritional lesions following prolonged exposure to cold (immersion foot) or severe cold (frostbite) may, in many instances, be prevented by suitable precautions on the part of those undergoing such risks, and much successful work has been done in improving conditions at sea, on land and in aircraft. When exposure is inevitable, every precaution has been taken, by the education of personnel,* to minimise its consequences. Moreover, as a result of experimental work and the collection and scrutiny of clinical records, the treatment of resulting lesions has been established on the broad biological principles indicated above ; and there has emerged a balanced conservatism which has been saving of both life and limb.

REFERENCES

Agote, L. (1915) *An. Inst. Clin. méd., B. Aires*, **1**, 25
Allen, F. M. (1941) *Amer. J. Surg.* **52**, 225
Blakemore, A. H., Lord, J. W. & Stefko, P. (1942) *Surgery*, **12**, 488
Butt, H. R., Allen, E. V. & Bollman, J. L. (1941) *Proc. Mayo Clin.* **16**, 388
Campbell, H. A. & Link, K. P. (1941) *J. biol. Chem.* **138**, 21
Charles, A. F. & Scott, D. A. (1933) *J. biol. Chem.* **102**, 425 & 431
Flosdorf, E. W. & Mudd, S. (1935) *J. Immunol.* **29**, 389
Greaves, R. I. N. & Adair, M. E. (1936) *J. Hyg., Camb.* **36**, 564
Howell, W. H. (1916) *Harvey Lect.* **12**, 272
Janský, J. (1907) *Sborn. klin. Praha*, **8**, 85
Kimpton, A. R. & Brown, J. H. (1913) *J. Amer. med. Ass.* **61**, 117
Landsteiner, K. (1900) *Zbl. Bakt.* **27**, 357
Landsteiner, K. (1901) *Wien. klin. Wschr.* **14**, 1132
Lewisohn, R. (1915) *Med. Rec., N.Y.* **87**, 141
Moss, W. L. (1910) *Johns Hopk. Hosp. Bull.* **21**, 63
Theis, F. V. (1933) *Surg. Gynec. Obstet.* **57**, 737

* [*see BMB* **403** *&* **404**]

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INJURY FROM EXPOSURE TO LOW TEMPERATURE : PATHOLOGY

W. BLACKWOOD, M.B., CH.B., F.R.C.S.ED.

When parts of the body are exposed to temperatures increasingly below that of their customary environment, they become " chilled." A tissue is said to be " chilled " when it has been damaged by exposure to a low temperature of a degree insufficient to cause frostbite. At first, normal physiological mechanisms are able to compensate for the cooling, but as the environmental temperature falls, and especially if loss of heat from the exposed part is accelerated by immersion in cold water or contact with cold metal, a level is reached when tissue damage occurs from which recovery is possible. Still lower levels of cold cause irrecoverable tissue changes. These temperature levels probably vary for the individual tissues of the part. Lewis (1942) has shown that recoverable damage may involve skin and subcutaneous tissues at a temperature from 18° to 15° C., and that irrecoverable damage by freezing occurs between − 5° C. and − 10° C. The fact that they do not become frozen at their true freezing point (about − 2° C.) is due to the ability of the skin to supercool.

The sequel to tissue damage is an inflammatory reaction or necrosis. The onset and rate of the inflammatory process depends upon the blood flow through the tissue ; it proceeds slowly while the tissue is still chilled, and is accelerated by warming. It is thought that inflammatory œdema causes circulatory obstruction, and so additional tissue damage. Repair follows in parts less seriously damaged ; dead tissues form sloughs.

Various degrees of chilling and differing environmental features give rise to different clinical manifestations with distinct clinical and histological features.

Immersion Foot

This is a condition in which the limbs are exposed for periods of about 22 hours or longer to moist conditions or to immersion in sea water, at temperatures from 10° C. to − 1·9° C. (Ungley & Blackwood, 1942). Damage of varying degree is caused to all the tissues of the limb. The damage to the skin is the most obvious, and results in blisters or gangrene. The damage to nerves, muscles and blood vessels is less obvious, but it is the cause of the prolonged disability from which the patients may suffer. The nerves show extensive demyelination of a degree and to a level depending upon the severity and extent of the exposure (Blackwood, 1944). All fibres are affected, especially the smaller myelinated and unmyelinated fibres. After rescue of the patient the nerves regenerate slowly, but perhaps incompletely. Incomplete regeneration is often associated with pain. This may be due to fibrosis of the superficial nerves (White, 1944), or to the transmission by incompletely regenerated nerves of impulses which cannot be normally synthetized and which are interpreted as pain.

In experimental animals the muscles show partial hyaline necrosis (Blackwood & Russell, 1943), followed by partial regeneration, and also by secondary degenerative changes due to denervation. Fibrosis of muscle is not a noticeable feature. The blood-vessels, which have been in spasm during exposure, dilate when the patient is warmed. In the experimental animal, they may show intimal swelling and damage to the media (Smith, Ritchie & Dawson, 1915) in the acute stage, but in man little evidence of this is to be seen in the later stages. Intravascular thrombosis is found only in veins and only near regions of necrosis or chronic infection. There is often a disturbance of the neuro-vascular mechanism, resulting in a state of cold-sensitivity, the pathological basis of which has not been demonstrated. Bones often show initial osteoporosis, followed by recalcification.

Frostbite

This condition may occur when portions of the body are exposed to temperatures below about − 5° C. Superficial tissues are rapidly chilled and there is crystallization of tissue fluids, rupture of cell walls, and death of tissue from the direct effect of the cold. When the exposure is of short duration, only the skin and superficial tissues are killed ; the deeper tissues remain intact because there has not been time for their cells to be much chilled. After longer exposures more tissue is irretrievably damaged. Stasis of blood occurs in the vessels, but thrombosis is not a feature of the exposure period (Greene, 1943).

Later an inflammatory response appears, with blistering,

PHOTOGRAPHS ILLUSTRATING THE PATHOLOGY OF IMMERSION FOOT

A. Human material

FIG. I—Dorsum of foot 50 days after exposure for 8 days on a Scottish hillside in cold and wet weather. On examination 6 days after the exposure, the hyperæmic stage of immersion foot was present. Both feet were similarly affected, with commencing gangrene in the toes. The great toe shown in this picture was subsequently amputated.

FIG. II—Lower extremities seen 4 months after a 10-day exposure in an open boat in the North Atlantic in November. The gangrenous feet had already been amputated when this photograph was taken. The wounds did not heal and there was severe pain. The legs were amputated below the knee.

FIG. III—Foot amputated 26 months after exposure for 10 days in an open boat. Before amputation the toes were missing and the skin was severely ulcerated, especially in the region of the os calcis, which was exposed. Same case as Figs. VIII and IX.

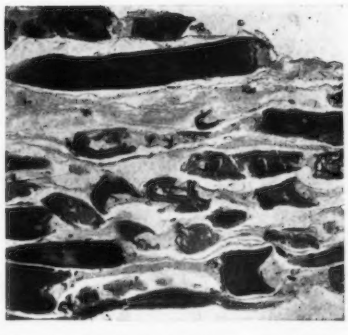


FIG. IV—Longitudinal section (× 90: hæmatoxylin and eosin) of extensor digitorum brevis muscle showing extensive muscular degeneration (Zenker's hyaline degeneration). Specimen from a man, exposed in an open boat for 34 hours in cold wet weather, who died half an hour before rescue. As judged from the condition of two survivors, he would have shown a moderate grade of immersion foot.

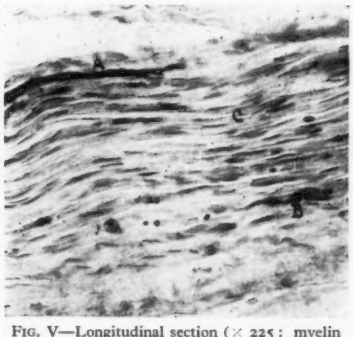


FIG. V—Longitudinal section (× 225: myelin stain) of interdigital nerve of toe, removed 10 weeks after exposure in an open boat for 2½ days. From a moderately severe case of immersion foot without gangrene. There has been severe degeneration, for only one normal myelin sheath (A) is seen. Globules of degenerate myelin are seen (B) and proliferated Schwann cells (C). There were sensory and motor changes in the lower limbs.

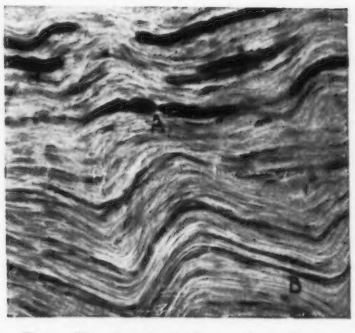


FIG. VI—Longitudinal section (× 225: myelin stain) of tibial nerve in lower third of leg, 10 months after exposure for 5 days in an open boat in severe weather. The patient suffered from a severe grade of immersion foot, and continued pain necessitated amputation of both legs. The section shows thick fibres (A) which escaped destruction and then regenerating fibres (B).

FIG. VII—Longitudinal section (× 265: axis cylinder stain) of thenar muscle of right hand 12 months after exposure in the sea for ½ an hour and in an open boat for 22½ hours in severe weather. Severe case without gangrene. The nerves are now returning to the muscle fibres which escaped destruction. A regenerating axon has grown out from its old neurilemma (A) and formed a nerve network (B) between the muscle fibres.

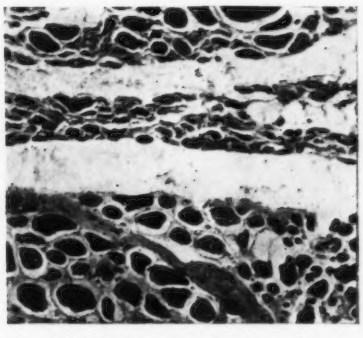


FIG. VIII—Transverse section (× 90: hæmatoxylin and eosin) of flexor hallucis longus of the foot shown in Fig. III. Muscle damage is unevenly distributed, as shown by the variation in calibre of muscle fibres.

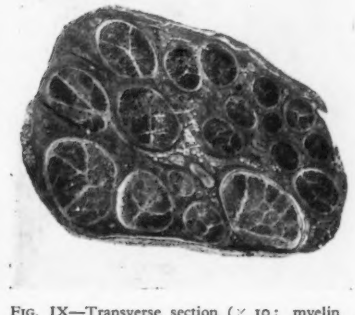


FIG. IX—Transverse section (× 10: myelin stain) of tibial nerve 6 cm. above extremity of lateral malleolus. Same case as Figs. III and VIII. The relative pallor of the nerve bundles indicates marked demyelinisation, persisting after 26 months.

[Figs. I–IX were first published in the paper reviewed in BMB **401** *and are reproduced by permission of the author and of the editor of the British Journal of Surgery.]*

PHOTOGRAPHS ILLUSTRATING THE PATHOLOGY OF IMMERSION FOOT

B. Animal experiments

The illustrations are all of tissues from rats' tails which had been immersed for 96 hours in artificial sea-water at 4°–5° C.

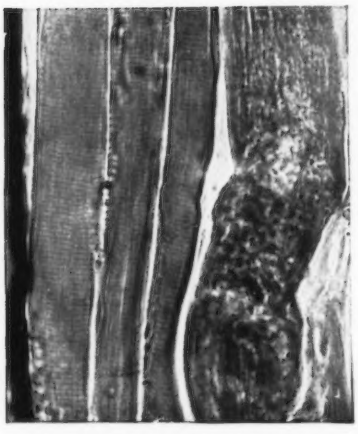


FIG. X—Longitudinal section (× 350: myelin stain) from rat killed immediately after exposure. The nerve on the left shows slight irregularity of contour. The muscle fibre on the right shows hyaline necrosis.

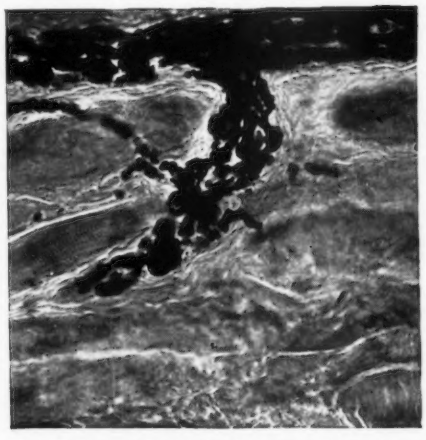


FIG. XI—Longitudinal section (× 350: myelin stain) taken from rat killed 3 days after exposure. The nerve fibres are degenerating, and many muscle fibres are degenerate.

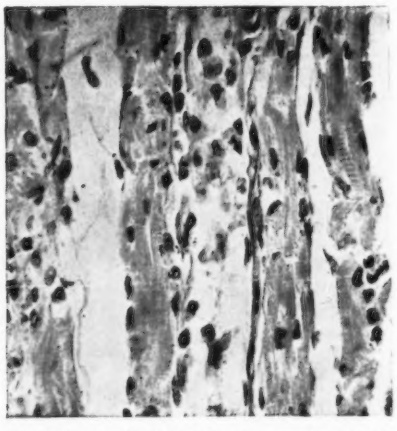


FIG. XII. Longitudinal section (× 350: hæmatoxylin and eosin) taken from rat killed 3 days after exposure. Marked cellular reaction in the damaged muscle fibres.

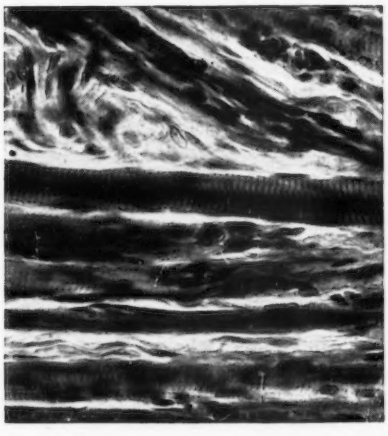


FIG. XIII—Longitudinal section (× 350: myelin stain) from rat killed 30 days after exposure. Increased cellularity of a muscle nerve, only one myelinated fibre remaining in the leash, which runs obliquely across the top of the picture. Shrunken muscle fibres are seen below, often with only a narrow strand of surviving myoplasm.

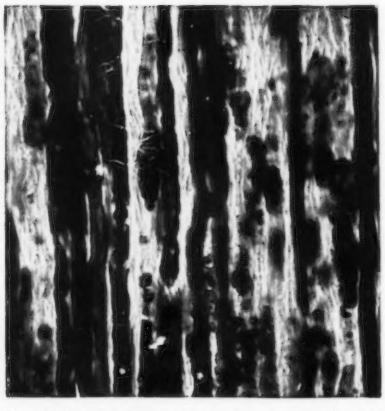


FIG. XIV—Longitudinal section (× 350: myelin stain) from rat killed 30 days after exposure. Main nerve of tail, showing numerous degenerate fibres.

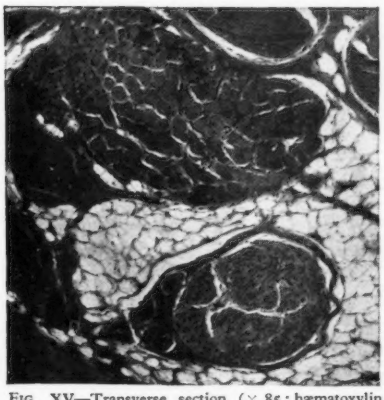
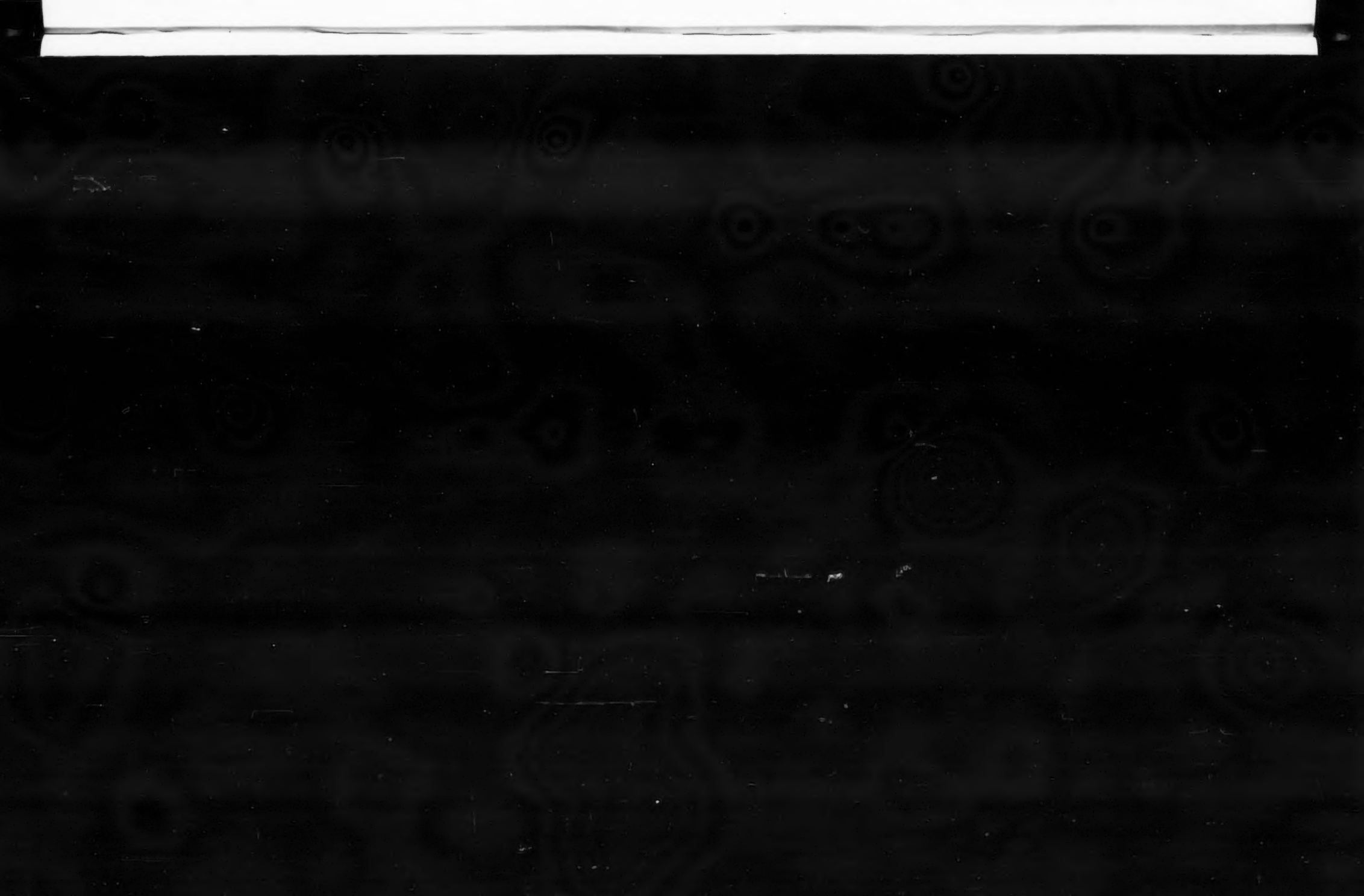


FIG. XV—Transverse section (× 85: hæmatoxylin and eosin) taken from rat killed 60 days after exposure. Changes are still present in muscle and nerve tissue.

[*Figs. X–XV were first published in the paper reviewed in BMB* **398** *and are reproduced by permission of the author and of the editor of the Edinburgh Medical Journal.*]



sloughing of dead skin or deeper tissues, and subsequent re-epithelialisation. Thrombi are now found in the arteries and veins of the skin and subcutaneous tissues, both in the necrotic regions and in those adjacent (Lewis, 1941). Obliterative endarteritis is reported to occur proximal to the necrotic area (Ducuing, d'Harcourt, Folch & Bofill, 1940, quoted by Bigelow, 1942). In frostbite, as in immersion foot, a state of vasoneuropathy and cold-sensitivity may develop after exposure. Detailed observations on the peripheral nerves do not seem to have been described. The bones show osteoporosis. Tissues affected by frostbite, like those damaged in immersion foot, are very liable to chronic pyogenic infection.

The pathological changes in high altitude frostbite have recently been reported by Davis, Scarff, Rogers & Dickinson (1943). In the hands the initial immediate response is constriction of the terminal portions of the superficial arterioles. Damage to the endothelium of the terminal cutaneous capillary loops results, after exposure, in transudation of fluid, or there may be thrombosis at the arteriolo-capillary junction. These changes produce large blisters, the line of separation being deep to the basal germinal layer of the epidermis; thus subsequent reformation of the epidermis is slow and derived from remnants of germinal epithelium persisting in the ducts of glands. When the exposure is sufficiently severe for the deeper arterioles to be injured, dry gangrene ensues. There is immediate loss of nerve function; and concentric intimal fibrosis of the digital arteries proximal to severe cutaneous lesions, and later prolonged vasoneuropathy have been recorded as sequels.

REFERENCES

Bigelow, W. G. (1942) *Canad. med. Ass. J.* **47,** 529

[1] Blackwood, W. (1944) *Brit. J. Surg.* **31,** 329

[2] Blackwood, W. & Russell, H. (1943) *Edinb. med. J.* **50,** 385

Davis, L., Scarff, J. E., Rogers, N. & Dickinson, M. (1943) *Surg. Gynec. Obstet.* **77,** 561

Ducuing, J., d'Harcourt, J., Folch, A. & Bofill, J. (1940) *J. Chir. Paris,* **55,** 385

[3] Greene, R. (1943) *J. Path. Bact.* **55,** 259

Lewis, T. (1941) *Brit. med. J.* **2,** 795, 837 & 869

[4] Lewis, T. (1942) *Clin. Sci.* **4,** 349

Smith, J. L., Ritchie, J. & Dawson, J. (1915) *J. Path. Bact.* **20,** 159

[5] Ungley, C. C. & Blackwood, W. (1942) *Lancet,* **2,** 447

White, J. C. (1944) *War Med.* **5,** 6

[1] [*see BMB* **401**] [2] [*see BMB* **398**] [3] [*see BMB* **402**] [4] [*see BMB* **396**] [5] [*see BMB* **397**]

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INJURY FROM EXPOSURE TO LOW TEMPERATURE: CLINICAL FEATURES, PREVENTION, TREATMENT

R. L. RICHARDS, M.B., CH.B.

The clinical features observed in an extremity after exposure to cold depend upon the duration and severity of the exposure. Brief exposure to severe cold results in freezing of tissues (frostbite); because they possess the property of supercooling, the temperature may fall to a level below tissue freezing point before frostbite occurs. A short, stinging pain may be felt; the affected area becomes hard, white and brittle, and remains so until thawing begins, when a red flush develops around it and gradually invades it. Blisters may form, and there is extravasation of serum and blood in the part, which leads to swelling and possibly to occlusion of its blood-vessels so that distal portions of an extremity, already endangered by freezing, become gangrenous. If infection is avoided, the end-result of severe frostbite is dry gangrene similar to that observed in severe forms of any type of peripheral vascular disease. Tissues immediately adjacent to the frostbitten area remain normal.

A more prolonged exposure to degrees of cold insufficient to cause freezing causes "chilling" of the tissues. This is the pathological basis of immersion foot, trench foot and allied conditions. During the period of exposure the distal parts of the affected extremities become numb, are usually white or yellowish in colour, and after a time begin to swell. When the extremities are removed from the cold environment, they pass through three consecutive phases which have been described as *prehyperæmic*, *hyperæmic* and *posthyperæmic* (Ungley & Blackwood, 1942). The *prehyperæmic* stage is a direct continuation of the clinical picture during exposure; the feet and legs are cold, swollen, pulseless, feel numb, exhibit objective loss of sensation of glove or stocking type, and motor activity in digits is impaired. Within hours, or in severe cases 1 to 2 days, a rapid change takes place to the *hyperæmic* stage; they become hot, flushed or pale, the pulses are full and bounding, and the patient complains of severe pain. The pain is usually of two types: (1) a severe burning aching pain felt in the hand or foot, and (2) short, shooting, lancinating pains which radiate into the digits; both types of pain are related to and aggravated by warmth, and are relieved by cold. In this *hyperæmic* stage there is every evidence of an exceedingly active circulation; if the extremity is dependent it becomes a deep reddish-purple, and on elevation it blanches almost instantaneously. Swelling increases, and blisters may form over pressure points and at the sites of unnoticed injuries sustained during exposure. Portions of the extremities destined to become gangrenous fail to warm, remain at first whitish or purple in colour, and finally become black and shrivelled. The area of objective sensory loss recedes towards the periphery; but remains of glove or slipper type, and there is anhidrosis of the soles or palms. The initial intense *hyperæmia* lasts for 7 to 10 days. Mild cases may pass from this to normality, but the majority exhibit a less intense hyperæmia lasting for 4 to 6 weeks. After this period the previously warm extremities show signs of vasomotor instability, and on occasion may be cool or cold. As time goes on the extremities tend to remain cold, and enter the *posthyperæmic* stage, in which they exhibit undue sensitivity to cold, which may take the form of attacks of the Raynaud phenomenon in the digits. Return of sensation is by nerve regeneration, and this is accompanied by the sensory phenomena which are associated with recovery from any nerve injury. When swelling subsides, wasting of the intrinsic muscles of the feet or hands becomes apparent, giving rise in the hand to an appearance like that of progressive muscular atrophy, and in the foot to clawing of the toes and a flat-footed springless gait. Another feature of this phase is hyperhidrosis, most marked in response to emotional or noxious stimuli. In the most severe cases gangrene occurs, with loss of digits and more rarely loss of hand or foot. Late sequels include persistence of a cold-sensitive state in the extremities, recurrence of pain when walking is resumed, deformities of the feet and absence of healing in the ulcers left under blisters or after removal of gangrenous tissue. Symptoms may persist for several years after exposure, and those who have been exposed to the effects of cold are more susceptible if re-exposure occurs.

High altitude frostbite recently described by Davis, Scarff, Rogers & Dickinson (1943) appears to occupy an intermediate position between acute frostbite and the syndrome resulting from chilling. A "wet type" associated with much œdema and blister formation, and a "dry type" corresponding to the classical picture of frostbite are described.

Prevention of frostbite is by adequate protection of the extremities, and this is usually possible under the conditions in which frostbite is likely to occur. Prevention of immersion foot is more difficult. The steps that may be taken have been fully outlined by the *Medical Research Council* (1943). Maintenance of the warmth of the body and avoidance of prolonged periods of immobility, of constriction of the limbs, and of injury to numb and swollen extremities are all of importance. Rubbing the feet with grease probably does more harm than good, but the wearing of stockings impregnated with vaseline is now advocated.

In the treatment of the established conditions, the aim is to avoid too rapid warming of the affected extremities. These should be elevated and exposed to the air in a cool environment; if a fan is available, it may be used to cool them; in hospital the use of cooling cabinets or icebags may be tried, but cooling should not be too intense, the aim being to maintain the temperature of the extremity in the neighbourhood

of 20° C. Once hyperæmia has developed, cooling is of definite value for the relief of pain. The direct application of heat has no place in the treatment of the chilled extremity. Indirect measures to increase the circulation such as reflex vasodilatation, vasodilator drugs, sympathetic block by local anæsthesia, and sympathectomy are of doubtful value; there is the danger that they may increase intravascular pressure and thus cause greater damage by increasing exudation. The extremities should be kept surgically clean; blisters are snipped with sterile precautions and raw areas are dressed with sulphanilamide and paraffined gauze. A prophylactic dose of anti-tetanic serum should be given. An attitude of surgical conservatism is called for; in the early stages the appearance of an extremity in which eventually only the tips of the digits will be lost may be most alarming. Amputations of the trimming variety are usually all that are required. In the late stages preganglionic sympathectomy may be of value in dealing with the cold-sensitive state.

REFERENCES

Davis, L., Scarff, J. E., Rogers, N. & Dickinson, M. (1943) *Surg. Gynec. Obstet.* **77**, 561

[1] Medical Research Council (1943) *M.R.C. War Memorandum No.* 8, London

[2] Ungley, C. C. & Blackwood, W. (1942) *Lancet*, **2**, 447

[1] [*see BMB* **403**]

[2] [*see BMB* **397**]

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TREATMENT OF ARTERIAL INJURIES

B. C. MAYBURY, M.B., B.S., F.R.C.S.

The treatment of arterial injuries in warfare is best considered under two headings: (*a*) the early treatment shortly after the infliction of the wound, and (*b*) the treatment of traumatic false aneurysm and the two varieties of arteriovenous fistula which may result from certain types of injury.

A. Early Treatment

Immediate control of hæmorrhage: In the forward battle area where only first aid is available, control is effected, in order of preference, by local pressure, application of forceps left *in situ*, ligature and, as a last resort, the application of a tourniquet; the last must be regarded as a confession of failure.

Treatment at centres where adequate facilities exist and where the patient can be retained: The treatment of the injury depends on the nature of the damage to the artery which may be: (i) complete division; (ii) partial division; (iii) contusion: (*a*) with thrombosis, (*b*) without thrombosis; (iv) arterial spasm.

i. *Complete Division*

The ideal of an axial anastomosis is rarely admissible in high-velocity wounds, as the damage to the severed ends by the "lateral concussive effect" of the projectile and the necessity for excising the damaged ends, results in a gap which makes impossible an anastomosis without tension; there is also the risk of infection which may lead to necrosis of the suture-line. This method may, however, be employed in stab wounds, e.g. by bayonet, in which there is no necessity to trim the severed ends and there is little or no risk of infection. To prevent thrombosis, the commonest cause of failure, heparin should be given intravenously, if other wounds in the same patient do not contra-indicate its use.

The use of an artificial cannula, the Tuffier's tube, or fine variety of Whipple's cannula, which is inserted in the gap and over which the severed ends of the artery are tied, provides a free flow of blood through the main artery for 3–4 days if heparin is given to prevent thrombosis. The cannula must be removed at the end of this period and both ends of the artery must be ligated. The time thus gained enables some degree of collateral circulation to be established and is valuable in reducing the risk of a rapidly spreading infection, anaerobic or aerobic, or ischæmic necrosis or both. The cannula should be of a slightly smaller diameter than that of the lumen of the artery to provide a stimulus for the developing collateral circulation.

A second variety of Whipple's cannula consists of two small metal tubes which unite a free vein-graft to the severed ends of the artery. The whole is intended to remain permanently *in situ*. Heparin should be given. This method should not be used in heavily contaminated wounds.

If the above methods are not practicable, both ends of the artery must be ligated, but often the limb has to be amputated within a few hours for a fulminating anaerobic infection or later for ischæmic gangrene. Among the limb arteries the popliteal, and the upper part of the posterior tibial, have the worst prognosis in this respect.

A primary amputation is the best procedure when there has been much muscular damage at the level of arterial severance, potential collateral vessels having been thereby destroyed.

ii. *Partial Division*

(*a*) *With small entry and/or exit wounds in the overlying soft tissues:* The imbrication of the various layers of muscle, fascia and skin across the track of the projectile prevents serious external hæmorrhage and the result is an arterial hæmatoma or early stage of false aneurysm, or if the vein has been wounded as well, an arteriovenous fistula. The diagnosis of this type of injury depends mainly on the presence of a short systolic murmur which is usually evident a few hours after the injury, although its appearance may occasionally be delayed. It is essential to recognise that the volume of blood flowing through the main artery is usually sufficient to cause no appreciable change clinically in the distal pulse and, nearly always, to ensure that there is no serious ischæmia of the limb. Although operations for these false aneurysms are eventually required, they are not often necessary in the first week, during which there is an adequate volume of blood flowing through the main artery and a progressive increase in the collateral circulation. By early operative interference the surgeon is therefore doing harm. These cases should be carefully watched, the limb should be immobilised and operation should be undertaken only if suppuration occurs, or if the pressure of the enlarging sac begins to interfere with the collateral circulation or with the flow through the main artery distal to its wound. Sometimes an incision through the deep fascia in the soft tissues away from the wound will relieve the pressure of an increasing hæmatoma and the distal pulse will return at once, though not always permanently.

(*b*) *With large entry and/or exit wounds in the overlying soft tissues:* As there is no soft-tissue barrier to the escape of blood externally, such cases do not often reach an operating centre without the previous application of forceps or ligature. Because he fails to realize that the presence of a distal pulse is not necessarily evidence that the artery is intact, and because he fails to auscultate the wound, a surgeon may proceed to excise a relatively small wound in the soft tissues, and may be confronted with a lateral wound in the main artery.

Suture of the arterial wound, as previously stated, is not often advisable; moreover, a longitudinal suture may cause so much narrowing of the lumen as almost or completely to obstruct the blood-flow. It may, however, be tried in stab wounds, especially if the artery is large and its wound small. Heparin should be given.

The method most likely to prove successful is to imitate Nature by making a barrier of soft tissues by suturing one or two layers of fascia or muscle over the artery and its wound, with the deliberate object of allowing a false aneurysm to develop and thereby maintaining a blood-flow through the main artery. Heparin should not be given.

The use of an artificial cannula, though technically easy, is not so effective, as the artery must be ligated not later than the fourth day. The artificial barrier method ensures the main arterial blood flow, usually for at least a week and often much longer.

iii. *Contusion*

(*a*) *With thrombosis:* Axial anastomosis is never possible owing to the length of the artery that must be excised.

An incision should be made through the normal vessel proximal to the clot, which is extracted, and the arterial wound is then sutured. A layer of muscle or fascia is sutured over the vessel to prevent external hæmorrhage if part of the arterial wall should subsequently necrose. Heparin should be given.

If the above procedure is not possible, an artificial tube should be used.

(*b*) *Without thrombosis :* This injury is not diagnosable clinically, but is discovered during the primary excision of the wound in the soft tissues. As the contused portion may slough and separate from about the fifth day, it is desirable to provide a barrier of muscle or fascia to ensure the formation of a false aneurysm and hence maintain the main arterial blood-flow, and also to prevent a serious external secondary hæmorrhage.

iv. *Arterial Spasm* *

Arterial spasm is not commonly produced by gun-shot wounds and is impossible to diagnose clinically from other arterial injuries which interrupt the main blood-flow.

If arterial spasm is seen during the course of the operation, the vessel should be gently washed with saline. Periarterial sympathectomy should not be done. If there is much damage to the prospective collateral vessels, the artery may be forcibly distended by the proximal injection of the vessel with saline. The condition often disappears spontaneously within 24 hours.

Treatment of the Ischæmic Limb

When the main arterial supply to a limb has been interrupted by ligature or injury, attempts should be made to reduce the demands on the limited blood available and if possible to increase the quantity and quality of blood.

The limb should be kept cool and at rest by wrapping in wool on a well padded splint and suspending it from a cradle from which ice-bags are also suspended. No pressure, either by bandage or splint, should be allowed to occur at the level of the collateral vessels. The limb should be elevated to prevent œdema, which would compress the capillaries.

Large transfusions of blood (in preference to plasma) should be given and oxygen should be administered by the B.L.B. mask. Warming the rest of the body by radiant heat or hot water bottles and immersing the unaffected limbs in hot water will increase the vascular dilatation of the affected limb.

The value of sympathetic block is still under discussion, but there is much clinical and experimental evidence, especially in the case of the lower limb, to show that sympathectomy increases the rate and volume of the collateral circulation. Although it is undesirable that a patient with a seriously wounded limb should be subjected to the major operation of lumbar sympathectomy, the same but temporary effect can be obtained by a local lumbar injection of procaine, or better, procaine in oil.

Measures should be taken to combat infection, which is a common and serious risk in an ischæmic limb when the wound is contaminated.

It is generally agreed that when the main artery has been tied, the main vein should be tied as well ; this procedure is, however, not universally accepted.

At a later stage, when natural warmth has returned to the limb and capillary return is normal, active muscular movements will help still further the collateral circulation.

B. Late Treatment : Traumatic False Aneurysm and Arteriovenous Fistula

The partial division of an artery, if treated expectantly, or by a musculo-fascial barrier, will inevitably lead to a traumatic false aneurysm ; or if the vein has been wounded as well, either to an aneurysmal varix or varicose aneurysm. Almost all false aneurysms are formed in this way, though a few may result from the rupture of a contused portion of the wall of the artery, the lumen of which has remained patent throughout.

i. *Traumatic False Aneurysm*

Operation is always required sooner or later, because the slowly but progressively enlarging sac will press on adjacent nerves, on the collateral circulation or on the distal portion of the main artery. The longer operation can be delayed, the greater will be the collateral circulation, the less the risk of ischæmia resulting from the usually necessary ligature, the easier its technical performance, and the less the risk of lighting up a dormant infection. On the other hand, the time of operation may have to be advanced owing to the appearance of one of the complications mentioned below. The time that should elapse between the date of the wound and the operation depends on many factors and varies greatly, but in general should be 2–4 months, and it should be longer in the case of the lower than the upper extremity. During the period of waiting, the muscles surrounding the sac should be kept at rest, but those of the distal portion of the limb should be given active movements to assist in the further development of the collateral circulation.

Although arterial suture may be attempted, especially of a small wound of a large artery, the operation is more difficult and not so certain in its results as the simpler one of ligature. Moreover, the longer operation is postponed, the less is the necessity for retaining the patency of the main vessel. In performing suture, arterial wall should be sutured to arterial wall, and no part of the false sac should be used to cover the opening. A double ligature is applied immediately proximal and distal to the arterial opening and any branch opening into the sac is also ligatured. Remote proximal ligature should not be done, as the aneurysm recurs and the second operation is much more difficult owing to the direct or indirect entry into the sac of several unknown and unmapped arteries.

For the temporary control of hæmorrhage, the use of a tourniquet, preferably pneumatic, is best. If this is impossible, the artery must be exposed above and below the sac, and either temporary clamps must be applied or tapes, tied gently but firmly with a single hitch over short lengths of rubber tube, must be used as ligatures. When the operation is completed the tapes are easily cut with a knife, the edge of which is directed towards the rubber. While it is important to obtain an adequate exposure of the artery and sac, it is also important to divide as few branches not connected with the sac and as little muscular tissue (which contains many arteries) as possible ; these vessels may be vital components of the collateral circulation. The sac is next freely incised and, while an assistant places a finger over the opening of any branch entering it, the surgeon working from inside the sac separates the artery from it for a short distance above and below the arterial opening. The artery is then ligatured immediately above and below the opening and divided between the ligatures. Any branches opening into the sac are similarly ligatured. Not only is there no necessity to remove the sac, but attempts to do so are difficult and may damage nerves incorporated in its wall and important components of the collateral circulation.

Occasionally, when immediate proximal ligature is impossible, a distal ligature may succeed if no other branches enter the sac or enter the vessel between the ligature and the sac.

Complications of traumatic false aneurysm : The treatment of pressure on the collateral vessels by an arterial hæmatoma in the early stages has already been mentioned under A (ii) above.

At a later stage internal hæmorrhage, as indicated by a sudden increase in size of the swelling, is caused by a leak at the junction of the attachment of the sac to the edges of the arterial wound. It is usually due to inadequate immobilization but may be due to sepsis. If due to the former it is treated expectantly provided the distal pulse is present but, if absent, by a separate fascial incision. If this fails the artery must be ligated. Suppuration sometimes occurs, usually during the second week, and its situation is at the junction of the clot with the surrounding tissues. It is treated by incision and ligation of the artery.

In the later stages, compression of adjacent structures, especially the vessels of the collateral circulation, may necessitate the advancement of the time of operation. Involvement of adjacent nerves is not of necessity an indication for interference ; when operation is eventually performed the nerve recovery is often very rapid. Œdema due to pressure on veins and lymphatics is treated by keeping the limb elevated.

ii. *Aneurysmal Varix and Varicose Aneurysm*

Operation for varicose aneurysm is always eventually required because of the progressively enlarging sac. However, as the rate of growth is slower than that of a traumatic false aneurysm, owing to the safety-valve effect of the opening into the vein, operation can usually be postponed for a longer period.

* [*see also BMB* 394]

Operation for aneurysmal varix may never be required, as the fistula tends to contract and, if small, may close spontaneously. The chief danger is the strain on the heart produced by the persistent tachycardia; rarely this may cause death within a few days, but usually the effects are not noticed for several months or even years. In general, therefore, all persistent aneurysmal varices should be operated on, but only after the lapse of some months or even longer if the tachycardia is not severe. The audibility to the patient of the bruit caused by arteriovenous fistulæ in the head and neck may affect his mental state and may necessitate the earlier performance of the operation. As other complications of arteriovenous aneurysm are uncommon, and of aneurysmal varix rare, early operation is not often necessary in the former and rarely in the latter.

During the early stages, the affected part must be immobilized and throughout there must be a reasonable amount of rest of the body to reduce the strain on the heart.

The remarks on arterial suture for the treatment of traumatic false aneurysm have a similar application to arteriovenous fistulæ. If used for the cure of an aneurysmal varix, the best approach is through the vein, the latter being subsequently ligatured.

The classical operation is the "quadruple ligature," i.e. immediate proximal and distal ligature of both artery and vein; in varicose aneurysm the sac must be opened and any branches opening into it must also be ligated. Proximal ligature of the artery alone must never be done; the limb would bleed to death through the fistula into the vein. Distal ligation of the artery alone would compel more blood than ever to enter the vein, and is therefore not permissible. In aneurysmal varix, the dense adhesions which bind the artery and vein together for a considerable distance above and below the fistula may make difficult the determination of the exact site of the fistula, which can be ascertained by noting the position where the murmur is loudest and of the highest pitch.

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TRAUMATIC ARTERIAL SPASM

SOL. M. COHEN, F.R.C.S.

Two main types of traumatic arterial spasm may be distinguished (i) the purely local response to direct trauma; (ii) a more widespread spasm, with minimal or no direct vascular trauma, but associated with other severe tissue damage, often local, which may give rise to shock.

i. Local Spasm following Direct Trauma

That the arterial wall contracts in response to an injury is well known, but the exact mechanism of this response is open to argument. It has been maintained (Leriche, 1939) that the local periarterial sympathetic plexus provides an intrinsic mechanism for such spasm; that the bruised vessel initiates a purely local "automatic" reflex, or one via the sympathetic ganglia. It has also been maintained (Leriche, 1937) that the damaged vessel "reflexly" maintains a state of spasm of the collateral vessels, and excision of a segment (arteriectomy) has been recommended as a necessary measure to abolish this collateral spasm. Nevertheless there appears to be reasonable doubt as to the existence of any such local reflex mechanisms.

This question is of more than academic importance, for the nature of treatment will depend on it. Anatomists (Woollard, 1926; Le Gros Clark, 1939) deny that any local arterial ganglion cells exist: no afferent sympathetic arc has ever been found, and the pathways for such reflexes are speculative. Clinical evidence is strongly against such a local mechanism. No reflex can explain why spasm should last sometimes minutes, sometimes hours, sometimes days. Procaine abolishes all nerve conduction; it should always and immediately abolish such "reflex" spasm if infiltrated around the vessel wall, but it does not. Bruising of vessels is common in the course of operations or injuries—yet persistent spasm is rare; the sutured vessel, after embolectomy or repair, will pulsate fully, despite the damage caused by tension of sutures.

For these reasons it has been urged (Cohen, 1941, 1944) that the key to the understanding of the peculiar and differing responses of vascular spasm lies in the appreciation of the natural contractile properties of the smooth muscle of the arterial wall. Smooth muscle responds only to a particular type of stimulus; this may be demonstrated in the bowel, which does not respond to cutting or pinching, but requires a definite type of stimulus—distension—to make it contract. Similarly with the vessel: a particular stimulus—a stretch-stimulus, or alteration in length of the muscle fibres—is required to initiate spasm. The local arterial bruise may thus be regarded not as an irritable focus, but as visible evidence that the vessel has sustained a severe blow.

In traumatic arterial spasm, the fibres of smooth muscle may decrease to ⅓ of their resting length, and the contracted vessel may appear as a mere thread. Smooth muscle needs little oxygen to maintain its new length, and the vessel can survive and maintain a state of spasm even whilst the tissues around are dead or dying (C. W. Clark, 1943).

Influence of the sympathetic: It may well be asked: what is the function of the interlacing periarterial sympathetic fibres? It would appear that the sympathetic system of the limb is best viewed as an effector mechanism—one of a number—for controlling the circulation. The sympathetic supply to the deeper vessels, compared with that to the skin and superficial structures, is relatively poor. The local vascular reflexes are usually concerned with the skin, but in states of emergency, such as shock or asphyxia, major vessel contraction may come into action.

The sympathetic system has been regarded as exercising a deleterious influence on the return of the collateral circulation, and the cold skin of the recovering circulation in the limb has been viewed as Nature's mistake. Of the two measures which have been advocated to release this "pathological" spasm of the collaterals—(*a*) arteriectomy—to remove an irritable local focus maintaining reflex spasm, (*b*) procaine block of the sympathetic ganglion—neither results in any sudden return of the circulation. The recovering circulation, as judged by the skin, still appears to creep slowly down the limb. It has been demonstrated that the cold pale skin may be associated with dilatation of the deep vessels supplying the muscles, and it is becoming increasingly recognised that the muscle circulation cannot be assessed from observation of the skin circulation alone (Friedlander, Silbert & Bierman, 1940; Grant & Pearson, 1938). The prime need of the recovering limb is for the early return of the circulation to the muscles; skin can survive long periods of ischæmia. Sympathectomy will not increase the blood supply to the muscles. The cold contracted skin of the ischæmic limb may be regarded as serving a useful purpose by preventing the accumulation of the limited blood supply in the skin vessels at the expense of the muscles.

Types of Traumatic Arterial Spasm

Several varieties of local spasm following the local vessel injury need separate discussion.

(*a*) *Spontaneous suppression of hæmorrhage:* Nature's method of sealing the divided vessel is indeed ingenious; the elasticity of the vessel, which speeds the blood flow, also initiates the impulse that seals the flow when the vessel is severed. The elastic tissue in a vessel is in a state of longitudinal stretch; to divide it is to alter suddenly the length of the muscle fibres and to initiate the muscle contraction. Only the muscle fibres adjacent to the point of division are thus affected, and this accounts for the very localised segment of vessel involved in such spasm. Where the recoil of the divided vessel is prevented, contraction is defective. Thus the partially divided vessel continues to bleed; a strip of adventitia or a shred of intact vessel wall will suffice to maintain the muscle length and thus prevent spasm. It is perhaps not sufficiently realised that forcible traction on a vessel, as in embolectomy or in dissection of an aneurysm, may induce prolonged and extensive spasm, especially in the distal vessel segment. This fact has an important practical application, for it emphasises the need for gentleness, and the avoidance of strong pull by artery forceps to deliver the vessel for ligature.

(*b*) *Arterial spasm following gunshot wound:* After a "near miss" by gunshot or bomb splinter, the vessel may be found in a state of spasm. This is often referred to as *stupeur artérielle* or sometimes as "Kroh's arterial spasm,"

following the description by French and German observers of cases in the 1914–18 war. Usually, it has been an accidental finding in the course of vessel exposure during wound excision; absence of pulse may have suggested it. Only a short segment of vessel may be involved. The spasm is rarely maximal, the lumen being merely narrowed and the distal limb is rarely seriously endangered. There is no evidence in these cases that the collateral circulation is also held in "reflex" spasm. It would appear reasonable to regard this form of spasm as consequent upon the sudden stretching of the vessel by lateral displacement due to the exploding force of the missile. Almost invariably, after inspection of the vessel and removal of adjacent blood clot without further intervention, such spasm disappears within 24 hours—with a satisfactory subsequent result.

(*c*) *Arterial spasm following fracture:* The importance of arterial spasm as a cause of gangrene or Volkmann's contracture has become increasingly recognised. Minor degrees of spasm are often found to follow fracture manipulations; the pulse disappears, as a rule only for a short period of 20–60 minutes, and there are no indications of nutritional or functional disturbance in the limb. The spasm may, however, last many hours and continue until the death of the limb. That the vessel is in spasm can be decided only by surgical exploration. It is important to remember that spasm is not the commonest cause of suppression of the pulse; often the vessel is divided, sometimes it is thrombosed, and at other times it is stretched over a bone spike, or is caught between the bone ends. It may even be compressed by an increasing hæmatoma.

It is tempting to wait and observe, for we know that such "spasm" often disappears spontaneously; but it cannot be assumed that this will happen, and where there is any doubt about the pulse, it is important to expose the vessel early. If the limb, and especially the muscles, are to be saved, operation must be done preferably within 6 hours of the injury. If the vessel is found to be in spasm, any surrounding blood clot should be gently removed and hot saline applied. Periarterial sympathetic stripping is inadvisable, and procaine injection locally or into the ganglia fails to release the spasm.

The degree of spasm varies; if it be mild, the vessel merely narrows and there are no distal ischæmic manifestations. In such cases, the vessel is best left alone, for the spasm will often disappear within a few hours. Plaster casts are contraindicated in such cases, as they may compress the collateral vessels over the bony points. Where the vessel is in very tight spasm—like a string—more desperate measures should be tried. Experience shows that this type of spasm does not relax spontaneously. Forcible stretching of the vessel lumen, by injection of saline under pressure through a No. 17 needle, is worthy of trial. It may be necessary to compress the vessel proximal to the needle point so as to raise sufficient pressure. Reasons for disbelief in the value of arteriectomy have already been mentioned above, and to do an arteriectomy where there is extensive collateral damage is to make certain of disaster.

A particularly obstinate type of vascular spasm sometimes follows fracture or injuries in the region of the popliteal fossa, particularly fractures of the condyles of the tibia, often with no displacement. Such spasm has also followed the use of the Böhler frame or other counter-pressure on the back of the lower end of the thigh. On exploration, some blood-staining of the adjacent areolar tissues may be seen, but there is no visible damage to the vessel. Often the patient has other injuries and has at some time been severely shocked. This type of spasm is distinct from the myogenic variety that we have been considering, and is probably best regarded as a shock response with local vascular emphasis. Its treatment will be considered in the discussion of the probably allied conditions, crushing injury and tourniquet spasm.

ii. Local Spasm without Direct Vascular Trauma

Under this heading may be considered the type of arterial spasm that follows the "crushing injury" or the tight tourniquet. In the cases of "crushing injury" that followed compression of limbs by fallen masonry (in some air-raid casualties) Bywaters, Belsey, McMichael, Dunn *et al.* (1942) found that an intense arterial spasm was the commonest cause of the limb ischæmia. There was no evidence of damage to the main arteries of the limb. The persistence of this spasm even after rapid liberation of the victim has been regarded as responsible for the subsequent muscle necrosis. According to Bywaters (1942), it is the autolysis of this dead muscle that determines the ultimate renal failure. A similar state of arterial spasm has occasionally been found to persist after tourniquet removal. Experimentally it has been found impossible to produce arterial spasm with the soft rubber tourniquet (Wilson & Roome, 1936), although Barnes & Trueta (1942) succeeded with a tight wire. The vessels were found uninjured, for the main artery of a limb, especially in the thigh, is well cushioned. Where the vessel in clinical tourniquet spasm has been exposed by operation, no vessel damage has been found. Further, this circumferential compression is not the type of stimulus that initiates arterial spasm, which requires a more brutal type of constriction.

The spasm appears to be best interpreted as a shock response, a reflex spasm, the reflex probably starting in the skin or deeper tissues and the sympathetic system serving as the efferent vasoconstrictor mechanism. It is probable that the reflex has to travel via the spinal cord, for experimentally the opposite limb vessel is also found involved in spasm. The appropriate treatment of this type of spasm is not yet decided. Hitherto, particularly in "crushing injuries," treatment has been concerned more with the saving of the life of the patient by attention to renal function, than with the limb. It had been hoped to prolong the vitality of the limb by keeping it cool, whilst waiting for the spasm to disappear spontaneously (Blalock & Duncan (1942) have shown experimentally that renal complications can be averted by cooling the limb). This type of spasm, however, appears to be particularly recalcitrant. Procaine injection of the major nerve trunks should be tried; both the afferent paths and the efferent sympathetic fibres will thus be blocked. Experimentally, sympathectomy has proved of definite worth.

General Condition and Spasm of Major Vessels

The generalised contracture of the smaller skin vessels in shock is well known, and is indicated by skin pallor. Studies of the effects of venesection (Barcroft, Edholm, McMichael & Sharpey-Schafer, 1944) show that with moderate hæmorrhage—800 cm.^{3}—the flow of blood to the muscles remains fairly constant unless syncope supervenes, when vasodilatation in the muscles has been demonstrated. With more severe hæmorrhage, the major vessels have been found, experimentally and clinically, to be small and contracted. In states of shock other than hæmorrhagic, arterial spasm has at times become sufficiently marked to obliterate the distal pulse, affecting different limbs in turn. Even in the mild shocked case, "irritability" of the vessel is a particular feature, and with the slightest dissection around it the vessel contracts; this may be of special significance in the type of vascular spasm that follows injury to neighbouring tissues. There is also some evidence that vasoconstrictive shock may determine the onset of a Volkmann lesion (Shepherd, 1943). This rationing of blood to the limbs when the general condition is poor, has to be carefully remembered in the treatment of the limb with vascular spasm. Attention to the general condition must therefore be the first consideration.

REFERENCES

Barcroft, H., Edholm, O. G., McMichael, J. & Sharpey-Schafer, E. P. (1944) *Lancet*, **1**, 489
Barnes, J. M. & Trueta, J. (1942) *Brit. J. Surg.* **30**, 74
Blalock, A. & Duncan, G. W. (1942) *Surg. Gynec. Obstet.* **75**, 401
Bywaters, E. G. L. (1942) *Brit. med. J.* **2**, 643
Bywaters, E. G. L., Belsey, R., McMichael, J., Dunn, J. S. *et al.* (1942) *Proc. roy. Soc. Med.* **35**, 321
Clark, C. W. (1943) *Brit. med. J.* **2**, 167
Clark, W. E. Le G. (1939) *The tissues of the body*, London, p. 189
Cohen, S. M. (1941) *Guy's Hosp. Rep.* **90**, 201
Cohen, S. M. (1944) *Lancet*, **1**, 1
Friedlander, M., Silbert, S. & Bierman, W. (1940) *Amer J. med. Sci.* **199**, 657
Grant, R. T. & Pearson, R. S. B. (1938) *Clin. Sci.* **3**, 119
Leriche, R. (1937) *Surg. Gynec. Obstet.* **64**, 149
Leriche, R. (1939) *Surgery of pain*, London, p. 244
Shepherd, J. A. (1943) *J. roy. nav. med. Serv.* **29**, 62
Wilson, H. & Roome, N. W. (1936) *Arch. Surg.* **32**, 334
Woollard, H. H. (1926) *Heart*, **13**, 319

REVIEW OF SELECTED PAPERS

Peripheral Vascular Injuries

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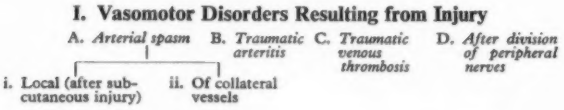
DISCUSSION ON IMMERSION INJURIES AND VASOMOTOR DISORDERS OF THE LIMBS IN WARTIME

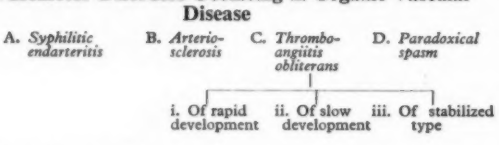
by J. R. Learmonth, C. C. Ungley, W. Blackwood, J. B. Gaylor, R. Greene & T. Lewis, *Proceedings of the Royal Society of Medicine*, **36**, 515–522, August 1943

Professor J. R. Learmonth said that in total war vasomotor disorders might affect all age-groups, and their effects were most serious in the elderly, in whom arteries had lost resilience. He suggested the following clinical classification:

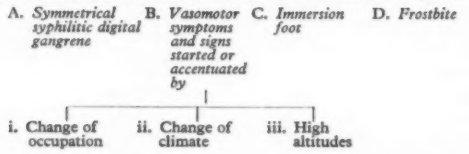
I. Vasomotor Disorders Resulting from Injury


A. Arterial spasm
B. Traumatic arteritis
C. Traumatic venous thrombosis
D. After division of peripheral nerves
i. Local (after subcutaneous injury)
ii. Of collateral vessels

II. Vasomotor Disorders Occurring in Organic Vascular Disease



III. Vasomotor Disorders from Local Effects of Cold


A. Symmetrical syphilitic digital gangrene
B. Vasomotor symptoms and signs started or accentuated by
C. Immersion foot
D. Frostbite
i. Change of occupation
ii. Change of climate
iii. High altitudes

Arterial spasm resulted from arterial injury; it was difficult to distinguish from arterial thrombosis. Both conditions might lead to vasoconstriction in collateral vessels. Spasm has been unaffected by periarterial sympathectomy proximal to the lesion, by attempts at reflex vasodilatation, and by preganglionic sympathectomy. The possibility of spasm or thrombosis was an urgent indication for exploration of the suspected artery, and excision of obviously injured segments. In the interval the nutrition of the limb should be conserved by avoiding the local application of heat, and possibly by the application of local cold. Uncomplicated arterial spasm recovered spontaneously.

Traumatic arteritis might occur in the main arteries of otherwise healthy young men, with resulting thrombosis. The clinical features were those of progressive ischæmia. These cases had been treated by preganglionic sympathectomy, followed by excision of the thrombosed segment of vessel in cases where this was feasible.

Traumatic venous thrombosis [or spasm] might give rise to reflex arterial constriction, which was relaxed by interrupting the appropriate preganglionic sympathetic fibres by paravertebral novocain injections.

In *organic vascular disease* the pathological diagnosis might be syphilis, arteriosclerosis, or thrombo-angiitis obliterans. The clinical features included both nutritional lesions and vasomotor attacks of the Raynaud type. In most cases treatment should be conservative; but in selected younger patients suffering from thrombo-angiitis, preganglionic sympathectomy might permit minor rather than major amputations.

Vasomotor disorders from local effects of cold included the rare condition of symmetrical syphilitic digital gangrene, and the numerically important group in which vasomotor symptoms and signs had been started or accentuated by change of occupation, change of climate, or exposure to the cold of high altitudes. Examples of the latter group were given. In general, operative treatment by preganglionic sympathectomy was employed only when it was desired to retain the patient in one of the Armed Services; otherwise treatment was conservative.

In *immersion foot*, first-aid should aim at preventing a too rapid return of blood flow to the chilled parts; patients were kept prone, by an open window, with the legs uncovered and elevated. In the later stages the attitude towards amputation should be rigidly conservative.

Surgeon Commander C. C. Ungley began by summarising the main clinical features of immersion foot, and described the three phases (prehyperæmic, hyperæmic and posthyperæmic) through which the extremities pass. Cases were classified into 4 groups according to the amount of sensory loss to cotton wool touches at 7–21 days after rescue:

i. Minimal cases with no, or at most transient, interference with nerve function.
ii. Mild cases with reversible nerve damage.
iii. Moderately severe cases with irreversible (degenerative) nerve lesions.
iv. Very severe cases with irreversible (degenerative) nerve lesions usually with gangrene.

A further subdivision into 9 smaller groups was also made. It was found that classification in this way correlated well with the severity of the other features of the condition, and a prognosis could be given for each group of cases.

Vasomotor observations in the hyperæmic and post-hyperæmic stages were recorded. Serial observations of skin temperature showed that in moderately severe cases the digits of the affected extremities might remain warm (30–35° C.) for as long as 7–8 weeks after rescue. During this period the normal skin temperature gradient from groin to toes was abolished. In the later stages of the hyperæmic phase the temperature of the toes became unstable and they might be very warm one day and cold the next. In the early stages a vasomotor paralysis was present and the temperature of the toes remained unaltered at a high level despite immersion of the arms in cold and hot water. Later, as the toes cooled, normal vasomotor activity might be restored or abnormal vasomotor responses might be observed. It was demonstrated that during the hyperæmic phase the cutaneous vessels of the feet were sensitive to physiological concentrations of circulating adrenaline released by insulin hypoglycæmia. Skin-temperature observations made during experiments in fan-cooling proved the efficacy of this method of treatment in reducing the temperature of the hyperæmic feet and also yielded some interesting and curious results. The speaker pointed out that there was a certain similarity between the findings in the hyperæmic stage of immersion foot and those which follow postganglionic sympathectomy. In the late hyperæmic stage it was often found that, if the feet were exposed to cold (e.g. by fan-cooling), their temperature would remain low for a very long period and would rise only very gradually despite efforts to accelerate warming by raising the body temperature. This phenomenon of delayed warming persisted into the posthyperæmic state, where it became associated with that of cold-sensitivity, and attacks of the Raynaud phenomenon might be observed. It was thought that these phenomena were not entirely vascular in origin but might be the result of partial denervation or re-innervation of cutaneous blood vessels whereby they become sensitised to adrenaline and similar substances and to the local vasoconstrictor effect of cold.

Dr. W. Blackwood dealt with the pathological findings in clinical and experimental immersion foot, which suggested that " at the time of immersion damage was done to all the structures in the limb of a degree and to a level dependent upon the length and severity of exposure." Changes were patchy; axis cylinders and myelin sheaths were killed, muscles were damaged. After rescue, nerves began to regenerate; muscles were either re-innervated or replaced by fibrous tissue. [See also experimental observations of Blackwood & Russell, 1943.]

Dr. J. B. Gaylor said that in the cold-sensitive stage of immersion foot there was pathological failure of reflex vasodilatation, varying from lack of response to a slow rise, even when the toes were anæsthetic.

Dr. Raymond Greene considered that the Continental opinion that the sympathetic supply to the affected parts should be interrupted was not physiologically sound. In immersion foot, cold had performed a postganglionic sympathectomy; and only in the cold-sensitive state, when partial regeneration had occurred, should preganglionic sympathectomy be considered. In the acute phase, increased

arterial input would do harm by increasing transudation. [See also Greene, 1943.]

Sir Thomas Lewis emphasised that all available evidence indicated that cold, as cold, damaged the tissues of immersed limbs, and that the damage was the greater the longer the exposure and the greater the degree of cold. [See also Lewis, 1942.] Sympathectomy should be reserved for the rare late *algid* phase of the affection. The hypothesis that arteriectomy would release reflex constriction of collateral vessels had not yet received physiological sanction or proof.

REFERENCES

[1] Blackwood, W. & Russell, H. (1943) *Edinb. med. J.* **50**, 385
[2] Greene, R. (1943) *J. Path. Bact.* **55**, 259
[3] Lewis, T. (1942) *Clin. Sci.* **4**, 349

[1] [*see BMB* 398] [2] [*see BMB* 402] [3] [*see BMB* 396]

Injury from Exposure to Low Temperature

396

SWELLING OF THE HUMAN LIMBS IN RESPONSE TO IMMERSION IN COLD WATER

by T. Lewis, *Clinical Science*, **4**, 349–360, December 1942

Sir Thomas Lewis, Director of the Department of Clinical Research, *University College* Hospital, London, adds in the present paper to his well-known work on the physiology of the skin. After noticing that his hand became swollen during long immersion in cold water he proceeded to analyse experimentally the causes of the swelling. Observations were made chiefly on the author's own hands. The volume of the hand was measured by a displacement method. Before each measurement the veins were emptied by raising the arm for one minute, after which the circulation was stopped by throwing a pressure of 200 mm. Hg into a pneumatic cuff embracing the elbow joint with the fist clenched.

Three factors can contribute to such swelling—imbibition of water by the skin, engorgement of the smaller blood vessels, and œdema. At 35° C. swelling can be entirely prevented by enclosing the hand in a loose rubber glove and keeping the arm horizontal. In the absence of these precautions swelling occurs, and is due partly to imbibition by the skin and partly to engorgement of the small vessels and œdema due to mechanical causes.

At 5° C. there is little or no imbibition of water by the skin, but swelling may amount to an increase of as much as 15 % which is more than twice that at 35° C. If the arm is kept horizontal swelling is only slightly diminished at the lower temperature. The swollen hand shows typical œdema and pitting of the subcutaneous tissues. The œdema fluid contains about 3 % of protein, showing that there is increased permeability of the vessel walls.

From the evidence presented in this paper the author concludes that long exposure to temperatures of about 15° C. or lower directly injures the tissues and leads to the liberation of "H-substance," the mechanism believed to be common to cutaneous injuries, and the production of an inflammatory œdema. The author points out that if 60 cm.[3] or more of albuminous fluid may be lost into a hand exposed to cold for 3 hours, it is clear that there must be a much greater loss of fluid and protein into the tissues when, for example, sailors are adrift in open and swamped boats, with larger areas of several limbs exposed for hours or even days. This factor should be taken into account in estimating the effects of exposure to cold upon the whole circulation.

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PERIPHERAL VASONEUROPATHY AFTER CHILLING: "Immersion Foot and Immersion Hand"

by C. C. Ungley & W. Blackwood, *Lancet*, **2**, 447–451, 17/10/42

This paper contains the first report on "immersion foot," a condition which was found to develop in men who had been exposed in open boats with their legs immersed in cold sea-water, and which closely resembled the "trench foot" of the 1914–18 war. The hands may be similarly affected. Investigations on some 80 cases seen in Scotland over a period of 2½ years are described. In these cases immersion in sea-water at −2° C. to 10° C. for periods up to 14 days resulted in damage to peripheral vessels, nerves, muscles and skin.

The affected limbs usually passed through three main stages,* *prehyperæmic*, *hyperæmic* and *posthyperæmic*. Late sequels included recurrence of pain, tingling, swelling or blisters, persistence of a cold-sensitive state or of hyperhidrosis, and occasionally circulatory defects suggestive of vascular occlusion. Of 18 moderately severe cases which were followed for 2 years, 7 were doing full duty, 2 had been invalided, and the rest were doing light duty such as office work at the end of that period.

The paper concludes with a detailed discussion of the pathogenesis and general principles of treatment of the condition.

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EXPERIMENTS IN THE STUDY OF IMMERSION FOOT

by W. Blackwood & H. Russell, *Edinburgh Medical Journal*, **50**, 385–398, July 1943

This important paper deals with the histological changes in muscle and nerve in the tails of rats, following prolonged immersion in cold (4°–5° C.) artificial sea water. Exposures of 48 hours produced slight lesions, of 96 hours marked lesions. The experiments were conducted in three series. In Series A the animals were allowed to recover slowly, spending the first 24 hours in an unheated (13·5° C.) room, before transference to the animal house (18° C.); in Series B, after removal from the cold water they were warmed rapidly for 30 minutes in shallow water at 29° C. before transference to the animal house; and in Series C they were heated for 3 hours in an incubator (37° C.) before transference.

In general, lesions were confined to muscle and nerve, being absent in skin, subcutaneous tissue, and blood vessels. In Series A, the histological findings in rats killed at varying intervals after exposure were as follows: In animals killed immediately after the longer exposures there was polychromic staining of the muscle fibres, loss of striation, and coagulation of segments of myoplasm into hyaline masses lying inside the muscle tubes (giving an appearance suggesting Zenker's necrosis). At 2 days after exposure, severely damaged myoplasm had disintegrated, the sarcolemma nuclei appeared to increase in number, and there was a general reaction manifested by capillary congestion, and polymorph and monocyte infiltration. After 3 days' exposure, reaction was well advanced, and the damaged muscle fibres were replaced by cellular tubes containing sarcolemma nuclei, phagocytes and some cells in mitosis. The destruction of fibres in any one muscle bundle was irregular. After 14 days, hyaline aggregations of myoplasm had been removed and the narrowed muscle tubes contained only a strand of striated myoplasm. Sarcolemma nuclei were numerous and were in groups. At 30 days, muscle fibres were still small, sarcolemma nuclei numerous and prominent. At 60 days, the picture was still much the same as at 30. The authors point out that fibre atrophy and sarcolemma nuclear increase may also follow denervation. Tower (1935) has shown that this change is not apparent earlier than four weeks after denervation.

Damage to the nerves also was apparent in animals killed immediately after the longest exposures, although its extent did not become clear as rapidly as with muscles. Irregularity of myelin sheaths with swelling of Schwann cells and occasional discontinuity of a few fibres was followed in 1–2 days by the early stages of Wallerian degeneration. At 3 days, the reaction was fully established and the disintegration of myelin and axis cylinder material was obvious. At 14 days, some of the myelin debris was removed, the Schwann tubes were easily identified, and increased cellularity of the nerve bundles was apparent. At 4 weeks, globules of degenerate myelin were still seen within the Schwann tubes in the main nerves, and the small branches to muscles showed a diminution in the number of the myelinated fibres.

In Series B the changes were fundamentally the same, but were more intense at 3 days. After 4 weeks nerve damage was similar, but there was less residual damage in muscle.

In Series C the muscle and nerve damage at 3 and 30 days was comparable to that in Series A.

* [*see BMB* 392]

The authors consider that neither denervation of muscle, nor organic vascular obstruction, contribute to the histological appearances, and that cold in itself may be responsible for the injury to muscles and nerves. The changes in muscle may be due to the selective inactivation of enzymes, those requiring most oxygen being retarded first; at the same time, slow oxidation continues, and metabolites accumulate. In muscle, this leads to the accumulation of lactic acid, and myosin, the main constituent of muscle protein, is unstable on the acid side of neutrality.

The authors point out that the similarity in nature and degree of the histological appearances in their three series does not support the hypothesis that slow warming limits the severity of the lesions. The article is illustrated by a number of superb photomicrographs.

[The authors' findings that warming after rescue did not lead to any alteration in the histological picture, should—as indeed they intended—be accepted only for the tail of the rat and for the range of temperatures obtaining in their experiments. They make the statement that 28° C. "cannot be considered a true warming up process;" and in Series C the temperature of the incubator was below the body-temperature of rats.

The clinical rule that in "immersion foot" the parts affected should not be locally heated (especially to temperatures above normal) is still valid.]

[*see also BMB* **394**: B. Animal experiments]

REFERENCE

Tower, S. S. (1935) *Amer. J. Anat.* **56**, 1

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IMMERSION FOOT AND IMMERSION HAND (PERIPHERAL VASONEUROPATHY AFTER CHILLING)

by C. C. Ungley, *Bulletin of War Medicine*, **4**, 61–65, October 1943

Immersion foot is a condition which follows prolonged exposure to degrees of cold insufficient to cause true frostbite. It may occur without actual immersion of the limbs, and the hands may be affected as well as the feet. Thus, as the author points out, the term "immersion foot" is a misnomer and "peripheral vasoneuropathy after chilling" is suggested as being more comprehensive and accurate. The condition is most often seen in shipwrecked mariners who spend long periods on rafts or in lifeboats with their feet almost continuously immersed, but it may be observed under any conditions in which extremities are exposed to cold and wet. The severity of the condition is found to depend chiefly upon duration of exposure and temperature. Other factors which may influence severity are clothing, particularly footwear (which affords some protection for short exposures but may later be dangerous if it constricts swollen limbs), immobility, sea-sickness, starvation, age and race.

During exposure, and for several hours after rescue, the affected extremities are cold, numb, swollen, pale, blue or mottled in appearance, and are pulseless (the prehyperæmic stage). Within a matter of hours there is an abrupt onset of hyperæmia. The extremities become hot and dry with full and bounding peripheral pulses. Colour-changes on elevation and dependency are striking, and indicate a very active cutaneous circulation. The amount of swelling usually increases during this phase, and blisters, petechiæ, ecchymoses and areas of superficial gangrene may appear. The appearance of the feet at this stage may be most alarming, but the amount of tissue loss is usually slight and deep gangrene is rare. Damage to peripheral nerves is manifest by subjective and objective sensory disorders, and by motor, vasomotor and sudomotor paralyses. Pain of a burning or shooting type may be severe and only relieved by morphine; in milder cases patients complain of tingling or pins and needles. These symptoms are related to warmth and dependency, and are alleviated by cooling and elevating the limbs. They increase in severity for the first 7–10 days after rescue and then gradually subside but may persist for as long as 6–10 weeks. Sensory loss of glove or slipper type for all varieties of sensation is often observed in severe cases. Hyperæsthesia is observed in milder cases, and, during the recovery phase, in cases with degenerative nerve lesions. Motor lesions consist of wasting, loss of power and impaired electrical reactions in the intrinsic muscles of hand or foot. Cutaneous vasodilatation, absence of reflex vasomotor activity on heating and cooling unaffected limbs, and failure of the skin temperature of the digits to rise after peripheral nerve block are evidences of vasomotor paralysis.

It is pointed out, however, that all the vascular disturbances in the hyperæmic stage of immersion foot cannot be accounted for on the basis of paralysis of sympathetic vasoconstrictor fibres. Tests of sudomotor activity in this stage show areas of anhidrosis approximately corresponding to those of sensory loss. The duration of the hyperæmic phase varies from a few hours to 14 weeks or more, depending upon the severity of the case. It is succeeded by a posthyperæmic phase in which the extremities are permanently cool or cold, hypersensitive to exposure to cold, and sweat excessively, particularly in response to emotional or painful stimuli. In some cases an intermediate stage is observed. In this the extremities, although usually warm, if exposed to a low temperature may become very cold and remain so for many hours, only becoming warm again very slowly (algid state). This condition passes into the cold-sensitivity of the posthyperæmic stage, in which attacks of the Raynaud phenomenon may occur. The physiology of these late vascular disorders is as yet imperfectly understood.

Cases are graded according to the amount of nerve damage as assessed by the amount of sensory loss: (*a*) minimal, with little or no interference with nerve function; (*b*) mild, with reversible nerve damage but no blisters or gangrene; (*c*) moderate, with irreversible (degenerative) nerve lesions; (*d*) very severe, with irreversible (degenerative) nerve lesions and gangrene.

It is found that classification in this way can be correlated with the severity of the other features of the condition and that the prognosis can be assessed for each group.

In material obtained from cases of immersion foot, pathological changes have been observed in muscles, nerves, arteries and veins. It is not certain whether these changes are the direct result of cold, or are produced indirectly by ischæmia or other factors.

In regard to prevention, the instructions given by the Medical Research Council (1943) are quoted. For the immediate treatment of immersion foot, elevation of the affected limbs, which should remain exposed to the air, and the application of warmth to the body are recommended. Heating the affected limbs is condemned. In hospital, dry-cooling the limbs by fans or ice-bags has given good results, particularly as regards the relief of pain. Rest in bed until all swelling has subsided and the patient can walk without pain is advocated. In the presence of gangrene, cleanliness and an attitude of surgical conservatism are required. Sympathectomy has no place in the early treatment of immersion foot, but may be of value for the symptoms in the late posthyperæmic phase.

REFERENCE

[1] Medical Research Council (1943) *War Memorandum No. 8: Guide to the preservation of life at sea after shipwreck*, London

[1] [*see BMB* **403**]

400

ÆTIOLOGY OF "IMMERSION FOOT"

by B. W. Goldstone & H. V. Corbett, *British Medical Journal*, **1**, 218–219, 12/2/44

The distinction between frostbite and chilling of the tissues is emphasised. The authors describe the clinical features seen in four parties of rescued men whose limbs were exposed to immersion in water above freezing point. A swollen stage and a stage of diminishing œdema are described. In the former the extremities are painless; in the latter there are severe pain, paræsthesiæ, tenderness of the feet with objective sensory loss; and the ankle jerks are stated to be diminished or absent. The resemblance of the picture to that of peripheral neuritis is stressed. Salt-water boils and duodenitis occurring in two cases (one fatal from hæmorrhage) are mentioned as complications.

It is considered that the clinical features of the immersion foot syndrome are the result of the combined effects of cold and relative ischæmia. In response to this double trauma the biologically weakest tissue, nerve, suffers first; hence the resemblance of the condition to peripheral neuritis. It is only if exposure is severe that the more resistant tissues suffer and gangrene results.

In contrast to current teaching, the authors believe that measures for active local vasodilatation have a definite place in the treatment of the syndrome, and state: "After the first few days of illness most of our patients were allowed a heat-cradle to their feet. When the pain became severe they begged for the cradle; it relieved the pain." They rightly stress the importance of the weakened neuro-muscular apparatus of the feet. Early weight-bearing is to be avoided and faradization and foot exercises are recommended.

[The authors do not appear to have differentiated between two distinct syndromes resulting from prolonged immersion of the extremities which have been described, the "peripheral vasoneuropathy after chilling" of Ungley & Blackwood (1942), and painful swollen feet secondary to prolonged dehydration and malnutrition (White, 1943). No details of the conditions of exposure are given, and it seems probable that many, if not all, of the present cases belong to the second group.]

REFERENCES

[1] Ungley, C. C. & Blackwood, W. (1942) *Lancet*, **2**, 447
White, J. C. (1943) *New Engl. J. Med.* **228**, 241

[1] [*see BMB* **397**]

401

STUDIES IN THE PATHOLOGY OF HUMAN 'IMMERSION FOOT'

by W. Blackwood, *British Journal of Surgery*, **31**, 329–350, April 1944

This is a histological study of material from 14 cases of immersion foot, consisting of a detailed and profusely illustrated account of the individual cases, with short clinical notes, followed by a consideration of the histological changes in the individual tissues.

The specimens varied in type from a digital nerve to a whole foot and leg; they varied also in the time which had elapsed between the period of exposure and the removal of the specimen; the earliest being a foot removed from a man who died during exposure, and the latest a lower limb amputated 26 months after exposure. In the later cases gangrene and chronic infection were sometimes present. Material was not obtained from cases during the early days following exposure.

"Blood vessels, muscles, nerve trunks and bone were particularly examined. The relative severity of injury to the tissues varied, being most severe in nerves and muscles." The proximal level of nerve and muscle injury was often far proximal to any areas of cutaneous necrosis.

Organic vascular changes were found to be minimal, except close to gangrenous extremities or in chronically infected stumps. There was no evidence that the cold-sensitive state, so often seen clinically, had a basis in organic arterial change.

Zenker's hyaline necrosis was seen in the foot muscles of a man who died during exposure. "Muscle was not seen again until 4 months after exposure. In the experimental animal (Blackwood & Russell, 1943) the early part of this interval was occupied by inflammatory changes in the necrotic muscle fibres, resulting in the production of slender hypernucleated fibres. It seems likely that a similar process occurs in man, but of this there is no histological proof." From 4 months onwards the changes were indistinguishable from those due to denervation, and were associated with severe degeneration of nerves to the muscles. From 12 months onwards there was evidence of re-innervation, and in some cases of restoration to normal. Fibrosis of muscle was not a prominent histological feature.

The nerves showed degeneration in all cases surviving exposure. In the later cases regeneration was present. Three cases allowed an estimation to be made of the rate of regeneration. This was slow but not unduly so. The nerve injury was often subtotal, and large myelinated fibres were amongst those surviving exposure. This feature, which was not specially noted by the author, suggests that the pathological process does not selectively attack the large myelinated fibres.

The bone: "In uninfected cases . . . there was new bone formation subperiosteally and around the haversian canals to a degree sufficient to reconstitute the radiographic normal."

The author has not discussed pathogenesis because of the lack of the very important evidence of very early post-exposure material. As stated in the preamble, "The histological findings in this series of specimens tell part of the story of the bodily reactions to severe chilling of the extremities. Though the story is incomplete, it seemed advisable to put the findings on record in view of their present interest and because no other account of the histology of human immersion foot has been found."

[*see also BMB* **394**: A. Human material]

REFERENCE

[1] Blackwood, W. & Russell, H. (1943) *Edinb. med. J.* **50**, 385

[1] [*see BMB* **398**]

402

THE IMMEDIATE VASCULAR CHANGES IN TRUE FROSTBITE

by R. Greene, *Journal of Pathology and Bacteriology*, **55**, 259–267, July 1943

The author reviews previous studies on the effects of cold on the tissues, and reports the results of his own experiments on mice. The tails of the mice were frozen by means of solid carbon dioxide, at temperatures from − 62° to − 67°C. Tails thus frozen for 15 minutes invariably became completely gangrenous, and were shed 7 days later. Freezing for 5 minutes produced similar changes but the proximal inch [about 2·5 cm.] of the tail survived. Freezing for 1 minute at − 62°C. was found to result in a constant sequence of events after the animal was removed from the freezing chamber; this was adopted as the procedure for most experiments, and is referred to as *standard frostbite*.

Macroscopic changes in standard frostbite. Immediately after freezing the tail is pale and hard; thawing is complete in 10 minutes and is associated with hyperæmia and slight œdema. No correlation was found between the amount of œdema at this stage and the ultimate result. Hyperæmia subsides in 5 hours; 24 hours later the terminal centimetre of the tail is brownish purple, gradually undergoes dry gangrene, and is shed at any time up to 18 days.

Microscopic changes. Apart from constriction of the caudal artery, vascular changes were confined to the portion of the tail destined to become gangrenous, in which swelling of the vascular endothelium and the formation within the lumen of the vessel of a shrunken pigmented mass consisting of clumps of erythrocytes were constantly observed. These changes were present 5 minutes after freezing. Later, there was further intimal damage and the development of signs of necrosis of the tissues of the tip of the tail, which continued up to the time of separation. In tails frozen for 2, 5 and 15 minutes the changes differed only in their intensity from those in standard frostbite. The author points out the significant fact that arterial thrombosis was not observed in any instance. The essential change is a loss of fluid through damaged vascular endothelium leading to an accumulation of erythrocytes in the blood vessels. True thrombosis may occur as a secondary phenomenon. These findings are in accordance with those observed by Rotnes & Kreyberg (1932).

The author's view is that little permanent damage is done to tissues as a result of exposure to degrees of cold less than that necessary to cause solidification, but that considerable damage may result from the events which occur during warming. In true frostbite, the tissues undoubtedly suffer damage from the direct effects of cold. The exact nature of the pathological changes which occur is imperfectly understood. From the point of view of treatment it is obviously desirable to have a clear picture of the changes which take place in the blood vessels of an extremity exposed to severe cold.

REFERENCE

Rotnes, P. L. & Kreyberg, L. (1932) *Acta path. microbiol. scand.*, Suppl. **11**, 162

403

A GUIDE TO THE PRESERVATION OF LIFE AT SEA AFTER SHIPWRECK

Medical Research Council: M.R.C. War Memorandum No. 8, London, 1943

In October 1941, the *Medical Research Council*, at the request of the medical department of the Royal Navy, appointed a

Committee on the Care of Shipwrecked Personnel. The task of the Committee was to examine conditions of exposure after shipwreck, with special reference to water requirements, extremes of temperature, food requirements and stimulants.

The Committee's recommendations are embodied in this Memorandum, which is written in simple language and is full of practical and authoritative information. Under "Lifeboat ailments," the essential features of frostbite and immersion foot and their prevention are summarized. The treatment of these conditions is outlined under "Treatment after rescue." Although this Memorandum is noted here because of its references to the effects of exposure to low temperatures, these references are only a small part of the subject matter.

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PREVENTION AND TREATMENT OF IMMERSION FOOT AND OF FROSTBITE

Ministry of War Transport, *Notice No. M.* 226, London

The following is the text of instructions in a leaflet issued to British mariners. It is recommended that all officers and men should read them carefully, and that copies should be kept in lifeboats.

Immersion Foot

When the feet or legs are immersed for many hours in water (fresh water, salt water or liquid mud), at low temperatures (temperatures measuring 15° C. (59° F.) or lower) the skin and underlying tissues very gradually suffer damage, which is the greater the colder the water and the longer the immersion. Sea water sufficiently cold to injure is usual in the Atlantic (winter and summer) from latitude 50° northwards.

Signs. Immersion in water cold enough to damage is painful, but this pain is not usually severe and soon lessens. In half an hour the immersed part of the limb is red and numb and power in it is reduced. This numbness and weakness get worse, and in 3 hours the limb is a little swollen. The swelling increases with time, especially if the limb is hanging down. All these early signs of damage quickly disappear if immersion ends and the limb can be warmed (warming must be very gradual—see "Treatment" below). Signs of damage are more persistent and more serious after immersion has lasted for several days; in such cases blisters or dark patches may appear on the skin and the skin may become broken. Only after long exposure in very cold water is recovery incomplete.

Prevention. Make all efforts to avoid those discomforts and disabilities which may lead to the risk of more permanent damage. The damage comes out of coldness, and the chief source of coldness in boats is cold water. Avoid contact with water as far as you can. Keep your feet out of water by keeping the bottom of the boat as dry as possible or by raising the feet. Even if you are wearing sea-boots the less water touches these boots the better; cold from the water will come through them. If your socks have become wet, wring them out, empty your boots and put them on again quickly; if you carry dry socks put these on rather than the damp ones. Try frequently moving your feet and toes. Unless the sun is out do not bare your feet to rub them; the exposure to the air will cool your feet more than the rubbing will warm them. If you rub them do it very gently. Rubbing the skin when it has become swollen, numb or tender, will do more harm than good. Greasing the feet is not much use. Keeping the upper parts of the body dry and warm is extremely important, because it also helps to keep the limbs warm. It is better to keep damp or wet clothes on the body under water-proof covering than to strip and wring out the clothes in a cold wind. Do not wear tight garters, boots or any tight clothing on the legs.

Treatment. Treatment will begin at once after rescue from boat or raft. Lift the patient on board; remove all clothes from the body, and wrap the body in warm blankets. Hot bottles may be placed against the body between the blankets, but none should be allowed to come near the affected limbs. These limbs should be handled only very gently and protected from the weight of bed clothes; and they should be kept cool, if necessary by exposure to the air; heating them in any way is harmful. The patient should be kept in bed with feet raised on pillows and cool until all swelling has gone, and subsequently until he is able to walk without pain, or until he comes under medical supervision. Blisters, open sores or darkened areas of skin should be dusted with sulphanilamide powder and kept covered by clean dry clothes, which are to be removed gently and renewed if they become stained; the part should be kept quite dry and no lotions, ointments or antiseptic except the powder above recommended applied.

Frostbite

True frostbite is a condition in which the skin and sometimes deeper tissue becomes actually frozen. The damage is not caused directly by cold, but by minute crystals of ice which form within the skin. Frostbite only occurs on land when the ground is hard frozen, or covered with dry snow or ice. At sea it occurs very rarely unless the sea water is freezing. Frostbite is almost unknown unless the temperature of the air is as low as — 10° C. (14° F.), and is very frequent only if the temperature is much lower (— 25° to — 30° C. or — 13° to — 22° F.). Wind encourages and sunlight discourages frostbite at a given temperature.

Frostbite affects uncovered parts of the skin; fingers, ears and nose when exposed are most prone to freeze and in this order. It affects parts that come in contact with very cold substances—the feet with ice or frozen ground, the fingers or mouth with cold metal.

Signs. When skin freezes it usually becomes pale, the colour is yellowish-white, it looks unusually opaque, it becomes of wooden hardness; this hardness is the only completely reliable and invariable sign of freezing. The patch of skin so affected is usually clearly marked off from the rest, both to sight and touch. The freezing usually happens in skin that is already tingling or numb with cold. The actual freezing occurs quite suddenly, sometimes after a sudden abrupt stinging pain or local pricking; it is also very apt to pass unnoticed. The frozen patch will enlarge if untreated, but the subject will not feel this. Skin once frozen remains quite unchanged until it thaws; it will then react by a more or less violent inflammation, according to the extent and hardness of the freezing.

Reaction. Directly after thawing the skin becomes bright red and hot. A short freeze is followed by itching and whealing of the skin, which disappears within a few hours; a severe freeze causes death of the skin which comes away after blackening and leaves a hole (or ulcer) in the skin; a very severe and extensive freeze will cause the death and consequent gangrene of the end of the limb affected (usually the toes).

Prevention. Frostbite is avoided by keeping warm. In intense cold the clothing should be windproof and abundant; thick gloves and ear protectors should be worn. Food should be abundant, fat and hot. The man should be in constant movement when out of doors; the feet especially should never be still for long. The socks should be thick and always dry. Cramped positions should be avoided. There should be no tight clothing on the limbs. Metals should not be touched with bare skin or with the mouth.

The feet and all exposed skin should be kept greasy, by gently smearing the parts with oil (as supplied in ships making Arctic voyages and in all life-boats and emergency life-rafts) or vaseline and by refraining from washing frequently.

Beard and moustache should be clipped short or shaved away; icicles will form on untrimmed beard from the moisture of the breath. When passing water the penis should be shielded from wind and afterwards carefully dried.

Men out of doors together should watch each others' faces for the first appearance of patches of frost; early freezing can be detected by the man himself by wrinkling the face to find areas of stiffness. Any frozen area should be thawed at once by placing a bare warm hand on it.

Treatment. Small frozen areas may be thawed rapidly by covering them with a warm hand; they require no subsequent treatment.

Larger and more severe freezes, as when a whole foot is involved, should be thawed by placing the limb inside a companion's clothes. Rubbing with snow is dangerous. If it is possible, the part should be thawed by immersing it in tepid or cooler water. On thawing, the skin will at once soften and will quickly become pink or red. If pain is severe on thawing, cool the part a little with water, or by short contacts with snow. It is important that after thawing warming up should be very gradual; application to the affected part of hot water or bottles or exposure before fire is dangerous.

Keep the part subsequently at absolute rest, the body well fed and warm, but the damaged part relatively cool. Put a light clean dressing, such as a wound dressing, over the damaged part and bandage very lightly; the object is as much to protect the part as to keep it clean. If there are any blisters or broken skin powder them with sulphanilamide powder before applying the dressing. Blisters should not be opened unless a pair of perfectly clean scissors is available, which should preferably be dipped in boiling water before use.

It is to be noted that in frostbite the amount of dead skin and tissue that eventually separates away is generally much less than at first might seem possible.

Treatment by Low Temperature

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COLD IN THE TREATMENT OF DAMAGE DUE TO COLD

by R. Greene, *Lancet*, **2**, 695–697, 12/12/42

The author of this paper, who is at present a physician in the *Emergency Medical Service*, has gained a special experience

of his subject in the course of expeditions to Mount Everest in which he has participated.

In true frostbite no treatment can save the affected tissue, the death of which is inevitable owing to blockage of the vessels by corpuscles left stranded therein by excessive transudation. On the other hand, in the state of physiological reaction in which tissues are responding normally to damage by cold, treatment is unnecessary. Frequently, however, as in immersion foot and trench foot, the tissues are merely chilled, in which state they are endangered by congestion resulting from a lesser degree of transudation than in true frost bite.

It is traditional that warmth greater than that of the human body is harmful in the treatment of the state of chilling. Smith, Ritchie & Dawson (1915) showed that, when damage by cold has occurred, water at only 37° C. causes rapid œdema or a rapid increase in œdema already present, an observation confirmed by Lewis & Love (1926). A previous investigation (1941) on mice by the present author demonstrated that frostbitten parts must be kept cool. Lake (1917) has shown that the survival rate of tissue-cultures is greatest between − 5° C. and + 5° C., while Lambert (1913) found that embryonic chick tissues survived best at + 5° C. Allen (1939) has shown that tissue necrosis after ligation of a limb is increased by heat and diminished by cold. In the author's opinion, all these findings lead to the conclusion that the frostbitten or chilled limb should be kept at a temperature just above freezing point, and he has accordingly designed a portable refrigerator (the Greene-Simpson Frostbite Cabinet), with the aim of testing the practical truth of this theory in the treatment of conditions such as immersion foot.

The cabinet consists of an upper and lower compartment. In the upper compartment is a metal tray in which two blocks of solid carbon dioxide (total weight 10–12 pounds) [about 4·5 to 5·5 kg.] are placed, each block wrapped in a separate canvas bag. The lower compartment opens in two sections, and has two padded apertures for the patient's feet, which are placed in position with sufficient cotton wool or bandage round the ankles to fill up the padded entrance holes when the cabinet front is replaced. If the cabinet is required for one limb only, the other hole must be completely plugged with cotton wool or bandage to prevent leakage of air. This lower compartment also contains a thermometer which registers on a dial at the back of the cabinet. Air circulates through two flues between the compartments. These flues are controlled by knobs at the back, by the manipulation of which the desired temperature can be maintained. When the refrigerant has evaporated to 2–3 pounds [about 1 to 1·5 kg.] it is removed from the bags and placed directly in the metal tray. The cabinet requires 8–10 pounds of refrigerant every 24 hours.

The author has not had the opportunity of fully testing the efficacy of this method of treatment, but suggests that, in the first instance, experiments should be controlled by treating only one limb in patients who are bilaterally afflicted.

REFERENCES

Allen, F. M. (1939) *Surg. Gynec. Obstet.* **68,** 1047
Greene, R. (1941) *Lancet*, **2,** 689
Lake, N. C. (1917) *Lancet*, **2,** 557
Lambert, R. A. (1913) *J. exp. Med.* **18,** 406
Lewis, T. & Love, W. S. (1926) *Heart*, **13,** 27
Smith, J. L., Ritchie, J. & Dawson, J. (1915) *J. Path. Bact.* **20,** 159

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TREATMENT OF IMMERSION FOOT BY DRY COOLING

by C. C. Ungley, *Lancet*, **1,** 681–682, 29/5/43

Since a previous report (Ungley & Blackwood, 1942), in which the condition was described in detail, the author has had the opportunity to try out methods of dry cooling in the hyperæmic stage of "immersion foot" in 2 men, A and B, exposed for 34 hours on a raft in the North Sea. In case A attempts were made to relieve pain on the 15th day after rescue by cooling the feet by means of a therapeutic refrigerator. This refrigerator was constructed on the lines described by Greene (1942), but made from wood instead of metal and not specially insulated. In spite of a good supply of solid CO_2 in the upper chamber and wide open flues, the temperature in the lower chamber of the refrigerator varied in accordance with fluctuations in room temperature, and the patient's left great toe, which had been cooled from 30° C. to 24° C. during the first 75 minutes, had warmed by the next morning to 25·5° C. and ultimately to 27·5° C. Pains were less frequent and less severe than on the previous night, but the patient experienced considerable discomfort through being unable to move his legs. For this reason it was decided that, although efficient cooling could doubtless be maintained by such a cooling cabinet if its walls were suitably insulated, cooling of the feet should now be attempted by means of an electric fan.

The patient was transferred on the 16th day to a cabin with a temperature of 16° C., and covered with blankets except for his feet. Exposed thus for an hour, the feet remained hot (great toes 31°–32° C.), pains were severe, and the feet often twitched. An electric fan at full speed 12 inches [about 30 cm.] from the soles cooled the toes in 2 minutes to 23·7° C., and in 80 minutes to 18·6° C. and 20° C. During the next 3 hours, with the fan at reduced speed and at a greater distance, the temperature of the great toes was maintained in the region of 17° C. and the dorsum of the feet at 22°–25° C. Pain and tingling ceased within 5 minutes of starting the fan, and returned whenever the fan was turned off and the feet became warm again. Cooling below 21° C. was liable to cause considerable discomfort and efforts were subsequently made to adjust the speed and distance of the fan so that the temperature of the feet was not reduced below 23° C.

In case B, during exposure of the unclothed body on the 13th day after rescue to a room temperature of 18° C. for 80 minutes, the left foot cooled satisfactorily, while the right remained hot (great toe 30·5° C.). The cold left foot showed a normal vasodilator response when heat was applied to the trunk. Aching and stabbing pains on the nights of the 14th and 15th days, chiefly in the right foot, were promptly relieved on the 16th day by cooling the right foot with a fan. From the 18th to the 23rd day the left foot cooled normally during exposure of the unclothed body to a room temperature of 17·1°–17·8° C. The right foot, however, cooled only under the influence of the fan, but did not immediately warm up again when the fan was switched off, which suggests that the skin vessels of this foot were beginning to regain tone. By the 27th day both feet cooled normally on exposure.

These observations confirm the findings of Webster, Woolhouse & Johnston (1942) that cooling the hot limbs relieves pain and tingling. In severe cases, however, Webster and his colleagues found that premature removal of ice-bags was followed by marked increase of œdema, extravasation of blood and spread of gangrene, clearly indicating the necessity, in such cases, for continuous cooling. In the present author's cases treatment was not begun until œdema had subsided, no exudation or necrosis followed interruption of cooling, and return to high skin temperatures was not necessarily associated with immediate return of pains and tingling. In such circumstances intermittent fan treatment in addition to exposure of the uncovered feet to room temperature would be used. Greene's refrigerator might, it is suggested, prove useful for severe cases requiring long uninterrupted cooling, and not intolerant to restriction of leg movements.

The author concludes that the mode of action of dry cooling requires further investigation, and points out that it has yet to be determined whether long cooling will influence the rate of recovery of the peripheral nerve lesions.

REFERENCES

[1] Greene, R. (1942) *Lancet*, **2,** 695
[2] Ungley, C. C. & Blackwood, W. (1942) *Lancet*, **2,** 447
Webster, D. R., Woolhouse, F. M. & Johnston, J. L. (1942) *J. Bone Jt. Surg.* **24,** 785

[1] [*see BMB* **405**] [2] [*see BMB* **397**]

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SOME USES FOR DRY COLD THERAPY, AND A PROPOSED COOLING CABINET

by W. G. Bigelow & E. C. G. Lanyon, *British Medical Journal*, **1,** 215–217, 12/2/44

The authors discuss the value of cold as a therapeutic measure and describe a form of cooling cabinet which may be used for its application. The cooling system consists of a cabinet which is cooled by cold brine circulating through coils in the

wall. The brine is cooled in a separate refrigerating machine and pumped to the cabinet. Several cabinets may be supplied from the one refrigerator, and it is claimed that the whole unit can be made in a mobile form suitable for the needs of an army in the field. The authors consider that in military surgery cold therapy may be of value in four sets of conditions :

i. *Frostbite and immersion foot.* Cooling of the extremities in the immediate post-exposure period is advised. Over a period of days the temperature of the cabinet is then gradually raised towards room temperature. The rationale of this treatment is the maintenance of a balance between local metabolism and the available blood supply.

ii. *Wounds of extremities with impaired blood supply.* The rationale of cold therapy is as above. Cold may also relieve pain and inhibit bacterial infection. Very low temperatures need not be used, and any tendency to vasospasm in the cooled limbs may be overcome by reflex vasodilatation, sympathetic block or vasodilator drugs.

iii. *Traumatic arterial spasm.* Refrigeration of the affected limb may be used as an adjunct to operative treatment (exposure of the affected vessel with or without arteriectomy), or as a form of conservative treatment to tide the limb over the critical period before a collateral circulation develops.

iv. *Peripheral embolism.* Cooling reduces tissue metabolism and allows the tissues to survive until the associated vasospasm is released and a collateral circulation is established.

Nervous Control of Peripheral Circulation

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THE MECHANISM OF THE VASOMOTOR REFLEXES PRODUCED BY STIMULATING MAMMALIAN SENSORY NERVES

by G. Gordon, *Journal of Physiology*, **102**, 95–107, June 1943

Stimulation of the cut central end of mixed nerves may produce a rise or a fall in general blood pressure according to the characteristics of the excitation. The author examined this phenomenon in decerebrate or anæsthetised cats, employing an electrical stimulus which could be quantitatively graded with respect to intensity, duration, and frequency of repetition. Full details of technique and results are given.

Using a stimulus (condenser discharge) with time-constant of one millisecond and frequency of three per second, the sign of the vasomotor response depends on the amplitude of the stimulus; peak amplitudes below 12 volts produce a blood-pressure fall, and amplitudes of the order of 28 volts produce a rise of blood pressure under the same conditions.

When the shock-duration is reduced to 0·1 millisecond, no pressor effect can be obtained even at 80 volts amplitude with frequencies below 100 per second.

There is, however, a tendency to produce pressor effects with frequencies of over 100 per second with shock configurations which at lower frequencies produce depressor reflexes only.

With certain stimulus-durations, therefore, either increase of amplitude or of frequency is found to reverse the vasomotor response, and the higher intensity or frequency is required for the pressor activity. The observations are altered in magnitude of response only, and not in kind, by section of the vagus or destruction of the nervous connections of the carotid sinus.

The author examined the peripheral mechanism of this differential sensitivity of the afferent side of the reflex by blocking the nerve proximal to the cut end with cocaine, cold, and asphyxia in turn.

He found that suitable doses of cocaine, which is known to act first upon the smallest fibres in a mixed nerve, abolish the pressor reflex while leaving the depressor activity; when asphyxia by arterial occlusion is applied to the preparation, the depressor reflex is abolished first, and may leave the pressure-raising action enhanced; asphyxia has been shown to act primarily upon the large diameter fibres. The effect of cold is similar to that of asphyxia.

The author concludes that the reflexes are mediated by different sets of afferent fibres, and that the pressor reflex with the characteristics of relative inexcitability to stimulation and ready inactivation by cocaine may be initiated through the C group of fibres.*

The author comments briefly on the possible importance of this differential vasomotor activity in connection with primary and secondary shock.

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NOTE ON THE SPINAL ORIGIN OF VASOCONSTRICTOR FIBRES TO THE ARM IN MAN

by J. R. Learmonth & R. L. Richards, *Quarterly Journal of Experimental Physiology*, **32**, 87–88, May 1943

In this brief communication the authors report, from the Department of Surgery of Edinburgh University, observations made on the vasomotor responses of the right hand of a male patient aged 19, in whom the anterior nerve roots on the right side were divided from the fifth cervical to the second thoracic segments inclusive. This operation was performed on 12/10/42 for the relief of gross athetosis of the right arm and shoulder due to a spastic right hemiplegia dating from birth.

" After operation the forequarter was completely paralysed with the possible exception of the rhomboids. It was not easy to be sure of this, because the shoulder girdle was still moved involuntarily, but much less violently, by the trapezius. Sensation was unimpaired save for some hypæsthesia in the second thoracic segment.

Vasomotor responses. On 25th November 1942 the patient was placed in a cool room. Thermocouples were attached to the left middle finger (control), and to the right little finger, right middle finger, and right thumb, and readings of skin temperature were taken at intervals of 3 minutes. At 23 minutes the feet and legs were immersed in water at 45° C. At 39 minutes the skin temperatures of the fingers began to rise, the rate of rise being nearly the same on both sides; the temperatures reached were somewhat higher on the left side. At 60 minutes the following temperatures were recorded on the right (paralysed) side: thumb 32° C., index 34° C., middle 34·5° C., ring 34° C., little 34° C. It was noted that the ulnar side of the right hand felt warmer than the radial side; the thermocouples were transferred to the thenar and hypothenar eminences, and to the wrists. The clinical impression was confirmed by the following readings:

	Right	Left
Thenar eminence . .	23·5° C.	32° C.
Hypothenar eminence . .	34° C.	34·5° C.
Wrist	24·5° C.	31° C.

The observations show that, in this patient, reflex vasomotor response in an arm was normal, when the most cranial source of its preganglionic vasoconstrictor fibres was the third thoracic segment of the spinal cord."

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VASOMOTOR DISTURBANCES IN THE HAND AFTER INJURIES OF PERIPHERAL NERVES

by R. L. Richards, *Edinburgh Medical Journal*, **50**, 449–468, August 1943

Cutaneous vasomotor disorders after injuries of peripheral nerves are in part due to the interruption of sympathetic vasomotor fibres and in part due to disuse and therm-anæsthesia as a result of division of motor and sensory fibres. Previous work has shown that all the effects of nerve division can be attributed to interruption of motor, sensory and autonomic fibres, and that there is no need to postulate a separate set of fibres which exert a " trophic " influence on the skin and its appendages. Sympathetic vasomotor fibres to the limbs have a predominantly peripheral distribution, and in the upper limb most of these fibres are in the median and ulnar nerves. Vasomotor disturbances are therefore pronounced

* [Erlanger, J. & Gasser, H. S. (*Amer. J. Physiol.* 1930, **92**, 43) classify the fibres of somatic nerves as belonging to groups A, B and C, according to their conductivity and irritability as demonstrated by the study of action potentials. C fibres have the slowest conduction rate.]

after division of these nerves, but are slight after division of cutaneous nerves to the more proximal parts of the limb or the radial nerve. In the present study, observations of vasomotor activity were made by recording skin temperature with thermocouples attached to a sensitive galvanometer. Only cases of complete division of the main nerve trunks of the arms are considered.

Immediately after nerve division there is a period of vasodilatation during which the denervated area of skin is hot, dry and flushed (the warm phase). This phase is attributed to the division of postganglionic vasoconstrictor neurons of the sympathetic system running in the peripheral nerves. The duration of this phase is variable, but is usually of the order of 21 days. It is succeeded by a phase in which the temperature of the denervated area is dependent upon that of its environment. Since the latter is usually low, this second phase may be referred to as the cold phase, and is permanent unless the divided nerve be repaired. The change from warm phase to cold phase is gradual and the factors responsible for the change are as yet imperfectly understood. It is believed that (*a*) sensitisation of denervated cutaneous blood vessels to circulating adrenalin, (*b*) degeneration of sensory fibres responsible for the local "axon reflex" (which according to Sir Thomas Lewis is so important in maintaining the temperature and nutrition of the normal digit), and (*c*) a lowered local metabolism in the denervated area are all important factors, but probably do not contain the full explanation of the later changes. The possibility that changes take place in the blood vessels, and particularly the arteriovenous anastomoses, cannot be excluded.

In addition to studying the resting vasomotor state of denervated areas, the author made observations on reflex vasomotor activity. He discusses the physiology of the vasodilatation and vasoconstriction which occur in the contralateral limbs when the legs or arms are warmed or cooled, and postulates that the afferent pathway for the reflex may be either through nervous channels or by the action of warmed blood upon a central thermo-sensitive vasomotor centre. On the efferent side the precise nature of the peripheral mechanism responsible for the rapid vasodilatation which takes place in a normal limb is not certain. Release of normal vasoconstrictor tone with opening of arteriovenous anastomoses is partly responsible, but it is suggested that the response may be initiated through vasodilator fibres.

In a limb which is the site of a peripheral nerve lesion, it is found that denervated digits show a profound disturbance of reflex vasomotor activity whether they are in the early warm phase or the later cold phase. A completely denervated digit will fail to show any response. A digit which is partly denervated (*e.g.* the ring finger in most lesions of the ulnar nerve) may show an almost normal response. The responses are rarely clearly defined, and no two lesions of the same nerve will give identical results. It is often found that a denervated digit which is adjacent to a normally innervated area (*e.g.* the middle finger in most lesions of the median nerve) may show a delayed and gradual rise in temperature. This is probably not due to true reflex vasomotor activity, but is an indirect result of the vasodilatation in the adjacent normal area. This raises local metabolism; vasodilator metabolites are produced, and, as the initial blood flow of the denervated digit is low, metabolites accumulate and are destroyed or removed slowly, thus causing delayed and gradual vasomotor responses. It is unlikely also that the nerve supply of deeper blood vessels corresponds to cutaneous sensory territories, and dilatation of these will result in a greater blood flow to digits adjacent to areas with normal innervation.

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REFLEX VASODILATATION IN SURGERY

by J. R. Learmonth, *Edinburgh Medical Journal*, **50**, 140–154, March 1943

The author of this paper, who is Professor of Surgery at Edinburgh University, points out that the amount of blood reaching the part is an important factor in the management of many surgical conditions.

The rate of flow of blood through a limb may be varied in accordance with (*a*) the general needs of the body, and (*b*) the local activity of the tissues. When one limb is heated by immersion in hot water, or by a hot air bath, heat is absorbed by the blood in the heated limb. Because of the local high temperature the metabolic rate of the tissues increases, thus further heating the blood through the limb. In order to maintain an even body temperature this extra heat has to be dissipated. This is effected by reflex vasodilatation of the vessels in the unheated part of the body, thus increasing the rate of blood flow in the unheated area.

The value of a method which can ensure a maximal blood supply in surgical conditions affecting the extremities needs no emphasis. These conditions fall naturally into 2 groups :

(i) *Infected wounds :* There is an obvious need for a maximal blood supply to promote rapid healing, to combat infection already present, and to prevent the spread of subsequent infection.

(ii) *Conditions in which the arterial channels are constricted or obstructed because of local vascular disease or dysfunction :* The need here is to ensure a blood supply adequate for the needs of the tissues, and thus to prevent tissue death.

There are two methods of using heat to increase blood supply : (*a*) local heating, and (*b*) reflex vasodilatation. In the first group of conditions, local heating can be used with impunity when the blood supply to the part is unimpaired. The local vasodilatation and the increased metabolic rate of the heated tissues induce a great increase in blood flow, because the arterial tree can dilate easily. In the second group of conditions, when the arterial tree is the seat of disease—*e.g.* arteriosclerosis—and the state of nutrition of the part is already precarious, local heating does not produce an increase in blood flow. As the local heating causes tissue metabolism to increase, the final state is worse because the tissues now become relatively ischæmic and local tissue death is brought nearer.

When reflex dilatation is used as a means of increasing blood supply in a diseased limb, there is no great increase in the tissue metabolism in the affected limb but the vessels will dilate to their maximum possible calibre. Local cooling of the affected limb is to be avoided (to prevent vasoconstriction) and equally the limb must not be allowed to create for itself a hot environment. If this were to occur, it would be equivalent to direct heating of the part. Frequent wiping to remove sweat, and the absence of dressings, help to conserve heat without overheating.

The method of reflex dilatation has been used with success in opening up collateral channels in cases of local injury to a main vessel (section, contusion, or spasm) or of thrombosis or embolism. In the sclerotic conditions of arteries the method can be employed with a measure of success which is dependent on the degree of sclerosis. It can often improve the blood flow sufficiently to avert or limit the extent of gangrene.

The condition of the nerves to the affected limb is of major importance. The final distribution of the vasoconstrictor nerves corresponds closely to that of the cutaneous nerves. Reflex vasodilatation cannot be effected in the area of supply of a divided peripheral nerve. Leriche (1939) has shown that a local lesion of a main artery (contusion or spasm) may impose a reflex vasoconstriction of its branches. Removal of the local lesion by excision is followed by an effective response to heating of a distant extremity. This effect is due to the interruption of the efferent vasoconstrictor nerve fibres when the artery is resected. Absence of response to reflex vasodilatation, in cases of wounding to an extremity in young individuals, justifies exploration of the wound to determine the state of the main vessel and, if this is found to be contused, excision of the affected segment.

The conditions in which reflex vasodilatation is of practical value are :

(1) In organic vascular disease in which gangrene threatens, and in patients who have had a limited removal of gangrenous tissue.

(2) In embolism of a main artery to secure, after initial spasm of the main vessel has disappeared, that collateral circulation will be maximal.

(3) After ligation for aneurysm to compensate for temporary circulatory deficiency. In the extremities, sympathectomy should precede operations on the aneurysm.

(4) In infective conditions of the hand or foot, after drainage has been established and fever is absent, to secure an extra blood supply for better heating and function.
(5) In ulcers of the extremities.
(6) After skin grafting, to increase the percentage of successful grafts.
(7) After tendon repair in hand wounds.

The response to the treatment may vary widely from patient to patient, but reflex vasodilatation in the above conditions will in some save life and in others a limb.

REFERENCE
Leriche, R. (1939) *The surgery of pain*, London

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SYMPATHECTOMY IN TREATMENT OF THE CRYOPATHIES

by E. D. Telford, *British Medical Journal*, **2**, 360, 18/9/43

The author states that in 5 cases he has treated the late results of injury from cold by preganglionic sympathectomy. Four of these had suffered from frostbite; one from immersion foot. In one case of frostbite, the patient was unable to work after 18 months because of chronic painful ulceration in the hands associated with Raynaud's phenomenon. After bilateral preganglionic section of the cervico-dorsal chain, the ulcers healed and the attacks of vasospasm were much reduced in frequency and severity. This patient has remained well. In another case, 8 months after suffering from immersion foot the patient was unable to walk and complained of constant burning pain in the feet, which were tender and sweated excessively. The toes had been partly shed and the stumps were ulcerated. After bilateral lumbar cord ganglionectomy the pain was immediately relieved and the ulcers healed rapidly.

In the lower limb, Telford prefers spinal anæsthesia as the pre-operative test in order to determine the possibility of vasodilatation, and states that if in such cases it produces a rise of 1° C., sympathectomy is justified. In the upper limb when all the fingers are affected he employs nerve block of the ulnar nerve at the elbow.

Ischaemia

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LOCALISED ARTERIAL THROMBOSIS OF INDETERMINATE ORIGIN

by J. R. Learmonth, W. Blackwood & R. L. Richards, *Edinburgh Medical Journal*, **51**, 1–20, January 1944

In this paper are described 4 cases of localised arterial thrombosis occurring in the limbs of otherwise healthy young adults. In one the brachial was the artery affected, in the other 3 the femoral. Trauma is considered to be the most likely precipitating factor, although in only one case was there a definite history of injury, and that at a site distant from the thrombosis; in the other 3 cases it is possible that an injury passed unnoticed.

The cases presented the cardinal features of ischæmia, still not readily enough recognised. Pain was a complaint of all 4, and a prominent feature of the brachial artery case in whom it was severe in the wrist and hand, and ultimately relieved only by morphia; it had been present for a year before nutritional lesions appeared. When the lower limbs were affected, the pain of intermittent claudication was experienced, and also distant pain in the peripheral territory of the blocked vessel, aggravated by exercise and felt severely at night. Other subjective symptoms were paræsthesiæ and a sensation of coldness of the extremity affected. Nutritional lesions (gangrene of an index finger and a perforating ulcer on the sole of the foot) had occurred in 2 cases before they came under observation; in the other 2, claudication and weakness of the affected limb were the presenting symptoms.

Diagnosis is dependent on clinical examination of the limb, the response to reflex vasodilatation, and arteriography. Clinical examination must include palpation of the peripheral pulses; in all cases these were absent distal to the site of the thrombosis, despite the presence of a good collateral circulation as demonstrated by arteriography. When the trunk or indifferent limbs are heated, the affected limb shows either complete absence or considerable limitation of reflex vasodilatation, compared with a normal response in the corresponding unaffected extremity. This is stated to be the result of (i) reduction of the available channels by which the resting blood flow of the extremity might be augmented, and (ii) vasoconstriction of collateral channels. The latter possibility is suggested because in 2 cases the amount of the vasodilatation obtained in the affected extremity by reflex measures was appreciably less than that subsequently obtained by preganglionic sympathectomy. Arteriography under spinal anæsthesia was performed on the cases in which the femoral artery was affected. It is of great value in determining the exact situation and extent of the thrombus and the state of the collateral circulation. Arteriograms demonstrate that in 2 cases there is a "filling defect" in the artery either beginning or ending abruptly. In the third case this clearly defined termination is not seen, but in this the arteriogram was made 2 years after preganglionic sympathectomy.

Pathological material was obtained in 2 cases (1 brachial and 1 femoral). The lumina of the affected vessels were found to be filled with well-organised and partly recanalised thrombus. The arterial walls showed damage chiefly to the intima, splitting of the internal elastic lamina, and slight focal damage in the media. The authors state that the damage to the elastica and media excluded thrombo-angiitis obliterans, and that the causative process was probably trauma. In the case where the brachial artery was thrombosed, there was marked crenation of the intima of the digital arteries, suggesting long-standing vasospasm.

The condition is differentiated from other forms of arteritis by the age and general good health of the patient and the fact that the abnormality is confined to one limb without any evidence of involvement of the vessels of the others. In all cases the Wassermann and Kahn reactions were negative.

Treatment consists of preganglionic sympathectomy and/or arteriectomy. One case remains relatively well 2 years after treatment by preganglionic sympathectomy alone. Arteriectomy may be called for under two sets of circumstances: (i) when there is evidence that the thrombosed segment of artery is reducing the available circulation by imposing spasm on collaterals, and (ii) when it appears likely that the thrombus may extend to block important and still patent collaterals. Preganglionic sympathectomy may be sufficient to deal with the former, the latter possibility can be determined only by arteriography, which may also demonstrate that arteriectomy would be difficult, if not impossible, without the sacrifice of a branch which is contributing substantially to the collateral circulation. Arteriectomy alone is insufficient; the patient so treated returned later having lost other fingers, and required preganglionic sympathectomy.

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A PATHOLOGIST LOOKS AT ISCHÆMIA

by W. Blackwood, *Edinburgh Medical Journal*, **51**, 131–143, March 1944

This is a short review of the common causes of ischæmia of the limbs, of the effects of this ischæmia and of the factors modifying these effects. Certain aspects are amplified by clinical descriptions, and appropriate histological material is illustrated by photographs. The causes of ischæmia are divided into those primarily due to (i) structural abnormalities, *i.e.* chronic arterial disease, thrombo-angiitis obliterans, traumatic arteritis, embolism and thrombosis; and those primarily due to (ii) functional abnormalities, *i.e.* the abnormal response of Raynaud's disease. The effects of ischæmia upon appearance and function are considered in relation to the pulse, skin temperature and colour, muscle power and nerve function; and the general structural changes and those in muscle and nerve are described.

Three cases are cited in which muscle biopsy was used to establish the diagnosis. One was a boy with a denervated ischæmic upper limb; the problem was whether the distal muscles were sufficiently alive for nerve suture to be worth while. The second was a case of persisting nerve and muscle

dysfunction following dislocation of the shoulder; the problem was whether this was due to nerve injury or to ischæmia. The third was a case of post-traumatic swelling of debatable origin in the thigh muscles, which was shown to be necrotic muscle. The second plate of illustrations is arranged so that the reader can readily distinguish the points of histological difference between denervated and severely ischæmic muscle.

The last section deals with the factors modifying the effects of ischæmia, *i.e.* the available blood supply, the metabolic demands of the tissues, the rate at which ischæmia develops and the duration of the ischæmia. The paper ends with a plea for the investigation of vascular problems by members of various departments working as a team.

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VOLKMANN'S ISCHÆMIC CONTRACTURE

by D. L. Griffiths, *British Journal of Surgery*, **28**, 239–260, October 1940

This comprehensive and authoritative paper is the text of a Hunterian Lecture delivered to the *Royal College of Surgeons of England*. Volkmann first reported in 1872 the rapid post-traumatic muscle contracture which subsequently became identified with his name. The condition is extremely rare, and the author shows that it developed in only 8 of 21,000 fracture cases receiving primary treatment in clinics directed by Professor Harry Platt of Manchester.

In a series of 32 cases encountered by the present author, the antecedent lesions were of the elbow (15), forearm (12), wrist (1), whole upper limb (1). The remaining 3 cases followed embolism of the brachial (1) and common femoral (2) arteries. The author briefly reviews the clinical features of the condition, emphasising the importance of cyanosis and swelling of the fingers as an early, though not invariable, sign. In severe cases absence of the radial pulse is probably a constant sign in the first 3–4 days. The author's description of the macroscopic and microscopic appearances of the affected muscle is illustrated by photographs and is consistent with previous accounts. Diagnosis in typical and established cases is easy, but in some cases there may be difficulty in distinguishing the condition from certain nerve palsies or muscle contractures after infected wounds.

The author reviews the literature on the pathogenesis of the condition, and discusses external compression, nerve injury, venous obstruction, arterial injury and embolism, the mechanism of the ischæmia, and the experimental production of a similar condition in animals. In a series of experiments on a small number of rabbits, he was unable to produce the Volkmann syndrome by the prolonged application of tourniquets to the hind limb (with or without ligature of the common femoral artery) or by ligature of the femoral artery with or without the vein (but without any tourniquet), or by any combination of ligatures applied to any or all of the external iliac, the common femoral, superficial femoral, and popliteal arteries. If, however, the common femoral and common iliac arteries on the same side were tied simultaneously, thus obstructing both main vessels and collaterals, a Volkmann type of contracture followed in 5 out of 10 rabbits. The remaining 5 failed to survive this operation.

On the basis of the clinical and experimental observations reported, the author concludes that Volkmann's ischæmic contracture is due to arterial injury and to the accompanying spasm of the arterial circulation. He stresses the importance of early preventive treatment, which must take the form of operative relief of arterial obstruction. Treatment of established contracture is also briefly discussed. On the whole, the prognosis is poor.

REFERENCE

Volkmann, R. von (1872) *Handbuch der Chirurgie* (Pitha-Billroth), 2, 846

416

TRAUMATIC ISCHÆMIA IN THE FOREARM AND LEG

by S. Stanford, *Lancet*, **1**, 462–463, 8/4/44

The author reports 2 cases of ischæmia following fractures. The first, a boy of 8, was admitted with a supracondylar fracture of the humerus. After an immediate reduction of the fracture, radiography showed redisplacement, and reduction was again undertaken with direct *x*-ray control. Some hours later, there was a median nerve palsy and absence of the radial pulse. The antecubital fossa was explored and the brachial artery was found to be white, contracted, and non-pulsatile. Blood-clots and several small fragments of bone were angulating and compressing the artery from behind. These were removed, without immediate improvement. Procaine (1 %) was injected into the adventitia of the brachial, radial, and ulnar arteries and periarterial stripping of the lower 3 inches [about 7·6 cm.] of the brachial artery was performed.

This was followed by immediate improvement, and there was ultimate good recovery, although biopsy of the forearm flexors 1 year later showed some muscular atrophy. The author suggests that the circulatory improvement at operation was " due just as much to the mechanical release of the bony obstruction as to release of the arteriospasm by stripping." *

The second case was of a man with a severe comminuted fracture of the tibia and fibula. After manipulation and application of a plaster case, there was evidence of defective circulation and the plaster was split. Some months later there was considerable muscular atrophy and impairment of circulation. There were gross trophic changes of the toes. The dorsalis pedis artery was just palpable, but no pulsation could be felt in the posterior tibial artery. Biopsy of calf muscle 18 months after the accident showed evidence of severe atrophy of muscle. Blood vessels and nerves appeared normal. The author refers to 6 cases of anæsthesia and muscle degeneration described by Parkes (1943), who suggested that tight plasters had interfered with the vascular supply of the nerve trunks and described the condition as " traumatic ischæmia of the nerve trunks." The present author suggests that his second case was also of this type, and regards the histological finding of muscle atrophy but no infarction as confirming this view.

REFERENCE

Parkes, A. (1943) *Lancet*, **2**, 15

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VOLKMANN'S ISCHÆMIA OF THE LEG

by M. Albert & W. R. D. Mitchell, *Lancet*, **1**, 519–522, 24/4/43

The authors describe 3 cases of injury to the lower extremity which they believe to have been followed by ischæmia of the Volkmann type of the muscles of the anterior tibial compartment. In 2 cases the injury had occurred in the war of 1914–1918, and there was in 1941 massive calcification in this region, as demonstrated radiologically and by surgical exploration. In the 3rd case, the injury was more recent (1941), and three months after the accident the anterior tibial compartment was occupied by a hard, painless swelling. This was found at operation to consist of dense fibrous tissue containing small blood vessels.

The authors give reasons for regarding the changes seen as due to a vascular injury, and not to direct injury of the affected muscles. They discuss in detail the theories which have been advanced by other writers in explanation of post-traumatic degeneration and subsequent calcification of muscle. They regard Volkmann's ischæmia as due either to temporary arterial obstruction or to venous occlusion. It is suggested that evidence of calcification may be available in old cases of this condition in the upper limb.

* [*compare BMB* 394]

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418/54

AIDS TO TROPICAL NURSING

by D. E. Cocker. London, Baillière, Tindall & Cox, 1944. 159 pages ; 12 illustrations. 4s. [£0.2]

This is one of several concise textbooks which have been designed to cover the field of knowledge required of the modern nurse. Nursing is the one branch of the medical services in the tropics which requires development more than any other. It is essential that the training of such nurses should be undertaken by those who have had actual experience of tropical nursing. This book has been written by the Sister Tutor of the *Presidency* General Hospital, Calcutta, and includes much more information on tropical diseases than is found in the average medical nursing books. It contains the following chapters : (i) keeping fit in the tropics—personal hygiene, inoculations, food and drink ; (ii) climatic disorders—glare, heat exhaustion, heat stroke, sun stroke, prickly heat ; (iii) deficiency diseases of the tropics—beriberi, pellagra ; (iv) nursing of tropical diseases ; (v) summary of communicable tropical diseases ; (vi) glossary. Chapter IV, treating of individual diseases, comprises the bulk of the book. There is an adequate index.

The information contained in this book is sufficient to fulfil the requirements of the examination syllabus of the General Nursing Council as far as tropical nursing is concerned.

The other books in this series deal with practical nursing, anatomy and physiology for nurses, hygiene for nurses, medicine for nurses, surgical nursing, gynæcological nursing, materia medica for nurses, fever nursing, tray and trolley setting.

418/55

GAS AND AIR ANALGESIA

by R. J. Minnitt. Second edition. London, Baillière, Tindall & Cox, 1944. 74 pages ; 18 illustrations. 5s. [£0.25]

Nitrous-oxide-air analgesia is a comparatively modern method for the relief of pain, and this little book gives a concise account of the technique of administration, with particular reference to the Minnitt apparatus. It is addressed principally to the midwife who will, under the supervision of the doctor, be required to use this method. The book is designed to diminish as far as possible the misfortunes so often consequent upon faulty technique. The specialist anæsthetist will also find it a useful basis for lectures to midwives, while the general practitioner will see in it a simple explanation of the part he expects the midwife to take in the administration of analgesia in obstetrics. Chapter headings : (i) history of the method ; (ii) apparatus and assembly ; (iii) technique of administration in midwifery ; (iv) contra-indications and causes of failure ; (v) statistics and legislative developments ; (vi) application to minor surgery. Appendixes : (i) self-administered analgesia for the midwifery of general practice : (ii) research notes.

418/56

ANTENATAL AND POSTNATAL CARE

by F. J. Browne. Fifth edition. London, J. & A. Churchill, Ltd., 1944. 622 pages ; 87 illustrations. £1 4s. [£1.2]

Professor Browne is a leading advocate in Britain of the importance of improved antenatal care as a prime factor in the reduction of maternal mortality and fœtal and neonatal morbidity. His book has grown much in size and reputation since it first appeared in 1935, and it is now the standard British work on the subject. The new edition includes an account of the Rh factor and erythroblastosis fœtalis, and of recent work on diet in pregnancy, especially in preventing various diseases and accidents of pregnancy.

Chapter headings : (i) history and development of antenatal care ; (ii) diagnosis of early pregnancy ; (iii) examination of the patient ; (iv) hygiene of pregnancy ; (v) influence of the emotions upon pregnancy and parturition ; (vi) constructive, educational and social aspects of antenatal care ; (vii) heredity ; (viii) maturity and post-maturity ; (ix) abnormal presentations and positions ; (x) multiple pregnancy ; (xi) abnormalities in the quantity of amniotic fluid ; (xii–xiv) hæmorrhage in early pregnancy ; (xv) unsuccessful pregnancy ; (xvi) the Rh factor and erythroblastosis ; (xvii–xviii) hæmorrhage in late pregnancy ; (xix) contracted pelvis and disproportion ; (xx) displacements of the uterus in pregnancy ; (xxi) vomiting in pregnancy ; (xxii) the toxæmias of late pregnancy ; (xxiii) diseases and disorders of the digestive system in pregnancy ; (xxiv) diseases of the circulatory system in pregnancy ; (xxv) diseases of the circulatory system ; (xxvi–xxviii) diseases of the nervous system in pregnancy ; (xxix–xxx) diseases of the ductless glands in pregnancy ; (xxxi) diseases of the respiratory system in pregnancy ; (xxxii) diseases of the urinary tract in pregnancy ; (xxxiii) affections of the skin in pregnancy ; (xxxiv) tumours complicating pregnancy, labour and the puerperium ; (xxxv) venereal diseases in pregnancy ; (xxxvi) the uses and value of radiology in obstetrics ; (xxxvii) postnatal care.

418/57

NUTRITION AND NATIONAL HEALTH

by Sir Robert McCarrison. London, Faber and Faber, Ltd., 1944. 75 pages ; 7 illustrations. 6s. [£0.3]

In 1936 Sir Robert McCarrison, formerly Director of Research on Nutrition in India, delivered three Cantor Lectures before the Royal Society of Arts. These lectures, which were published in pamphlet form, aroused considerable interest. They have for long been out of print and their re-publication in book form at a time when world-wide attention is being directed to the important part played by food and nutrition in health, is especially to be commended.

The three lectures expound the author's argument that properly constituted food is the greatest single factor in the promotion of good health and faulty food the greatest single factor in the production of bad health. McCarrison has performed experimental work of fundamental importance in the field of nutrition. He has produced peptic ulcer, urinary calculi, and many avitaminoses by faulty feeding of animals. Much of this work has been carried out in India, where the profound effect of food on physical efficiency is strikingly exemplified. These lectures provide experimental proof of the fundamental importance of food in relation to health. They are entitled : (i) food, nutrition, and health ; (ii) relation of certain food essentials to structure and functions of the body; (iii) national health and nutrition.

418/58

TEXTBOOK OF SURGICAL TREATMENT

including Operative Surgery

edited by C. F. W. Illingworth. Second edition. Edinburgh, E. & S. Livingstone, Ltd., 1944. 564 pages ; 230 illustrations. £1 10s. [£1.5]

Nineteen specialists have co-operated in the production of this textbook, under the editorship of the Regius Professor of Surgery in the University of Glasgow, and the book reflects the considerable personal experience of these contributors. As the editor points out, surgical treatment implies not only the actual surgical operation, but also many ancillary measures of preparation and after-treatment. It is devoted primarily to full accounts of pre- and post-operative measures ; major operations and highly specialised procedures are dealt with in more general terms. In its first (1943) edition, the book was well received. This second edition has been revised to incorporate the most recent developments in surgical treatment ; the chapter on burns has been rewritten, and some new illustrations have been included. The book attains a high standard of production, is well illustrated and adequately indexed.

Chapter headings : (i) pre-operative and post-operative care ; (ii) wounds and wound infections ; (iii) the treatment of burns ; (iv) amputations ; (v) affections of blood vessels ; (vi) affections of the skull and brain ; (vii) affections of the spine and spinal cord ; (viii) peripheral nerve injuries ; (ix) surgery of the autonomic nervous system ; (x) diseases of bones ; (xi) diseases of joints ; (xii) tuberculosis of bones and joints ; (xiii) paralysis and contracture ; (xiv) fractures ; (xv) affections of the shoulder region and arm ; (xvi) affections of the elbow ; (xvii) affections of the hand ; (xviii) affections of the hip ; (xix) affections of the knee ; (xx) affections of the foot ; (xxi) radiotherapy ; (xxii) plastic surgery ; (xxiii) affections of the face, mouth, jaws and salivary glands ; (xxiv) affections of the neck ; (xxv) affections of the thyroid gland ; (xxvi) affections of the larynx, pharynx and œsophagus ; (xxvii) affections of the breast ; (xxviii) affections of the thorax ; (xxix) hernia ; (xxx) affections of the stomach and duodenum ; (xxxi) affections of the intestines ; (xxxii) affections of the rectum and anus ; (xxxiii) affections of biliary tract, pancreas, and spleen ; (xxxiv) affections of the appendix ; (xxxv) affections of the kidney and ureter ; (xxxvi) affections of the bladder and urethra ; (xxxvii) affections of the male genital tract.

418/59

FORWARD SURGERY IN MODERN WAR

by W. H. Ogilvie. London, Butterworth & Co., 1944. 96 pages ; 12 illustrations. 10s. 6d. [£0.525]

This gives a concise account of the primary treatment of battle casualties and is based on three years' surgical experience in Africa and the Middle East, where the author, a distinguished surgeon, served as consultant to the East Africa Force and afterwards to the Middle East Force. The book is intended for the general surgeon in one of the services who is already trained in operative technique but new to the surgery of wounds encountered in modern warfare. The chapters are entitled : (i) general principles of forward surgery ; (ii) wounds ; (iii) resuscitation ; (iv) the surgical treatment of wounds ; (v) *x*-rays: immobilization: evacuation ; (vi)

wounds involving bones and joints; (vii) arterial injuries: amputations; (viii) abdominal wounds; (ix) chest wounds; (x) wounds of the head and face; (xi) burns; (xii) an outline of treatment at the base. The book is well arranged typographically; the illustrations consist of clear line drawings. There is no index, which is perhaps not a serious omission in a book of this size.

418/60

SURGERY OF MODERN WARFARE

edited by Hamilton Bailey. Sub-editor for medicine, C. Allan Birch. Third edition. Edinburgh, E. & S. Livingstone, Ltd., 1944. Part VI: 212 pages; 146 illustrations. 15s. [£0.75]

With this part, the third edition of *Surgery of Modern Warfare* is completed. Parts IV–V were reviewed in BMB 387/46. In Part VI are discussed (i) wounds of the large intestine; (ii) wounds of the rectum and buttocks; (iii) post-operative abdominal complications; (iv) wounds and injuries of the kidneys; (v) wounds of the bladder; (vi) wounds of the urethra; (vii) wounds of the scrotum, testicles and penis; (viii) heat exhaustion and other effects of heat; (ix) subtropical surgery; (x) the stretcher case; (xi) wounds in naval action; (xii) an outline of the medical services of the British army; (xiii) the transportation of wounded; (xiv) hospital organization in the Emergency Medical Service; (xv) the organization of a first-aid post. In an appendix of 30 pages Hamilton Bailey supplies some details omitted by the other contributors and reviews the literature which has appeared during the preparation of the book. Finally, a detailed index completes this work, which must be regarded as an outstanding contribution to the literature on war surgery.

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BACTERIOLOGY FOR MEDICAL STUDENTS

by A. D. Gardner. Third edition. Oxford University Press; London, Humphrey Milford, 1944. 264 pages; 31 illustrations. 8s. 6d. [£0.425]

Bacteriology is a vast subject, and it is proper that the beginner should not too soon be overwhelmed with facts. This is a short readable account of the subject, confined to the needs of the student and general practitioner and omitting details of technique. It is thus well suited to serve as an introductory manual. The new edition includes an account of the most recent advances in immunology, disinfection, and chemotherapy, while the rest of the book has been carefully revised and the section on ultramicroscopic pathogens augmented.

Chapter headings: (i) microbes and their significance to man; (ii) form and structure of bacteria; (iii) cultivation of bacteria; (iv) pathogenic action; (v) the pyogenic cocci: *Staphylococcus*; *Streptococcus; Neisseria*; (vi) *Bacterium* (intestinal infections, etc.); (vii) *Bacterium, Proteus*; *Pseudomona*; *Vibrio* (cholera); (viii) *Pasteurella* (plague); *Brucella* (Mediterranean and undulant fever); *Hæmophilus* (whooping-cough, etc.); (ix) *Corynebacterium* (diphtheria); (x) *Mycobacterium* (tuberculosis, leprosy); *Actinomyces* (actinomycosis); (xi) spore-bearing rods, *Bacillus* (anthrax); *Clostridium* (tetanus, gas-gangrene, botulism); (xii) spirochætes: *Treponema* (syphilis, etc.); *Leptospira* (infectious); (xiii) Protozoa (tropical diseases); fungi (skin infections); bacteria of minor importance; (xiv) ultramicroscopic agents: viruses; bacteriophage and border-line organisms; (xv) the defences of the body: immunity; hypersensitiveness; anaphylaxis; (xvi) the prevention of infection: sterilization; disinfection; antiseptics; chemotherapy.

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PRACTICAL ANÆSTHETICS

for Students, Hospital Residents and Practitioners

by J. Ross Mackenzie. London, Baillière, Tindall & Cox, 1944. 63 pages; 136 illustrations. 10s. 6d. [£0.525]

The object of this manual is best stated in the words of the author, who intends it "to form a foundation on which the medical student and the medical or surgical hospital resident may build the practice of anæsthesia and analgesia and as a guide to the practitioner or the occasional anæsthetist, who desires to know the type of anæsthesia best suited to his patient in various circumstances." The practical aspect of the subject predominates in the descriptions of the anæsthetic agents and the methods of their administration. The recognition of anæsthesia as a speciality and the institution of a diploma in anæsthetics have in recent years raised the standard and status of anæsthetic practice in Britain. Chapter headings are: (i) preparation of the patient; (ii) premedication and basal narcosis; (iii) inhalation anæsthesia; (iv–v) the inhalation anæsthetic agents; (vi) the Nuffield Oxford ether vaporiser No. 1, rectal oil-ether anæsthesia, ethyl chloride anæsthesia; (vii–viii) the gas anæsthetic agents; (ix) endotracheal anæsthesia, trichlorethylene; (x) intravenous anæsthesia; (xi) local and regional analgesia; (xii) analgesia and anæsthesia in obstetrics; (xiii–xiv) complications and sequelæ of anæsthesia; (xv–xvi) the choice of the anæsthetic agent; (xvii) essential anæsthetic equipment with descriptive notes; (xviii) anæsthesia in infants and children; (xix) oxygen therapy and helium therapy; (xx) anæsthetics—to-day and to-morrow.

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SOCIAL SERVICE IN A GENERAL HOSPITAL

by D. Manchée. London, Baillière, Tindall & Cox, 1944. 164 pages. 6s. [£0.3]

It is almost 50 years since the first hospital almoner in Britain started work at the *Royal Free* Hospital, London, her task being to ascertain the individual circumstances of each patient and to assist those whose home or financial circumstances were likely to prevent the patient from benefiting by hospital treatment. From this has sprung the hospital almoner of the present day, the "dispenser of the hospital's charitable services," whose medico-social work, carried out in close co-operation with the medical and nursing staff of the hospital, includes the collection of information regarding the patient's home and industrial life, help in alleviating factors likely to retard his progress to renewed health and a normal life; the provision of surgical and other appliances; the arrangement of convalescent and other institutional treatment; and district nursing, health visitors, and other aspects of after-care. Too often patients are ignorant of the services at their disposal. The hospital almoner thus has an important part to play in the field of social medicine.

Miss Manchée, who is Almoner at *St. Mary's* Hospital, London, and who therefore writes with practical knowledge of her subject, describes in detail the hospital social services in Britain at the present time. She has come to know the problems of the patient, both in peace and war, and her book is interspersed with many anecdotes, both sad and gay, which illustrate the varied nature of the work of the almoner. This book is important as being the first full account of medico-social work in the hospital; its publication coincides with the desire of many to learn more of this aspect of treatment and with the promise of a great post-war expansion of social medicine.

Chapter headings: (i) the purpose of hospital social service; (ii) the growth of industrialism; (iii) diagnosis and treatment of social distress; (iv) the expectant mother; (v) the sick child; (vi) care of the cancer patient; (vii) the diabetic; (viii) the ophthalmic department; (ix) the medical patient; (x) the orthopædic; (xi) the almoner in casualty; (xii) needs of the air-raid casualty; (xiii) the clinic for venereal diseases; (xiv) convalescence; (xv) meeting the cost; (xvi) interviewing; (xvii) the almoner student; (xviii) functions of the almoner's department; (xix) equipment of the almoner's department.

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THE BLOOD PRESSURE AND ITS DISORDERS INCLUDING ANGINA PECTORIS

by J. Plesch. London, Baillière, Tindall & Cox, 1944. 149 pages; 57 illustrations. 15s. [£0.75]

In this book, which Dr. Plesch describes as a companion volume to his larger work, *The physiology and pathology of the heart and bloodvessels*, an attempt is made to apply in practice the results of the experimental and theoretical investigations which the author has made of the blood pressure by means of the tonoscillograph. The tonoscillograph, of which the author is such a keen advocate, permits of the recording, as well as of the determination, of the arterial blood pressure, and has therefore the advantage over other methods of recording the blood pressure of providing a permanent record which can always be available for comparison with subsequent records. In addition it is claimed that it provides information concerning the state of the arterial wall. After demonstrating, with copious diagrams and equations, what is meant by the effective pressure, the maximum pressure, the minimum pressure, and the pressure amplitude, the book proceeds to deal with the clinical application of these observations, paying particular attention to angina pectoris, which is considered to be "a disease of the whole circulation," there being "no known fact which would justify the assumption that angina pectoris is specifically a heart disease." In addition to supplying a full exposition of the author's views on this question, treatment is detailed at considerable length. The book also includes sections on venous pressure and œdema.

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THE DIAGNOSIS AND TREATMENT OF DELINQUENCY

Clinical Report on the Work of the Institute for the Scientific Treatment of Delinquency during the five years 1937 to 1941

by E. Glover. Published by the Institute, 17 Manchester Square, London, W. 1. 32 pages.

The Institute for the Scientific Treatment of Delinquency was founded in 1932, having for its object the provision of facilities for examination and treatment of anti-social conduct, particularly among young people. At first, physical examinations took place in premises lent by the *West End* Hospital for Nervous Diseases, but in 1937 the Institute opened a clinic with adequate equipment for examination, diagnosis and treatment of out-patient delinquents. In addition, the Institute has been active in the education of public opinion, the provision of training facilities for students and the organisation of centres outside London. It has not confined itself exclusively to the psychological or sociological interpretation of delinquency, but has developed a plan of research to cover the diagnostic and therapeutic fields.

This Report shows that 719 cases were referred to the Institute between 1937 and 1941. This clinical material is analysed as to sources, diagnosis, recommendations, disposal, psychological treatment, results, and after-history, and the Report concludes with a note on possible future developments in the diagnosis and treatment of delinquency.

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